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BULLETIN

OF

THE JOHNS HOPKINS HOSPITAL

Vol. XIII.—No. 134.] BALTIMORE, MAY, 1902. [Price, 15 Cents.

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THE EFFECT OF CERTAIN POISONS ON INORGANIC FERMENTS.

By Harry C. Jones, Ph. D.

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A brief review of the work of Bredig and his pupils on Inorganic Ferments has already appeared in this Bulletin.[1] This work has been continued and some striking relations between the inorganic ferments and certain enzymes have been established.

The inorganic ferments in question are finely divided metals, such as platinum, gold, silver, cadmium, etc. The metals are obtained in the finely divided condition by dipping bars of the metal in question into pure water, and passing a heavy electric current from one bar to the other through the water. The metal under these conditions is torn off in a state of such fine division that it remains suspended in the water in the form of a solution. Indeed, the metal is in such a state of division that the most powerful microscope fails to reveal any heterogeneity when a drop of the solution is brought beneath it. From this it might be concluded that these solutions of the metals are true solutions. Such, however, is not the case. True solutions freeze lower than the pure solvent, have less vapor-tension or boil higher than the pure solvent, and exert osmotic pressure. The solutions in question of the metals have none of these properties. They have the same freezing-point, the same vapor-tension or boiling-point as the pure solvent, and do not exert any osmotic pressure. To distinguish these solutions of the metals from true solutions they have been termed *pseudo-solutions* or *colloidal solutions.*

These are in no sense a new class of solutions. Examples of colloidal solutions have long been known. Solutions of starch, albumen and the like are as truly colloidal solutions as those of the metals in question. The solutions of the metals, however, have certain remarkable properties which seem to resemble in many respects the properties of the organic ferments. Some of these were referred to in the earlier review. It was noted that a colloidal solution of platinum accelerates the oxidation of alcohol to acetic acid in the presence of the oxygen of the air, as well as the organic ferment *mycoderma aceti.*

Finely divided iridium decomposes calcium formate into calcium carbonate, carbon dioxide and hydrogen in the same manner as certain bacteria.

[1] The Johns Hopkins Hospital Bulletin No. 114, September 1900, p. 224.

The analogy between the action of the finely divided metals and ferments was not limited to fermentation proper, oxidation, etc.; but held also for diastatic phenomena such as the inversion of cane sugar. The finely divided metals effected the inversion of cane sugar in a manner which was strictly analogous to the action of invertase.

Again, hydrogen dioxide was decomposed into water and oxygen in exactly the same manner by the colloidal solutions of the metals as by the ferments in the blood.

Many relations similar to the above were pointed out by Bredig and von Berneck,[2] in the first communication on this subject. These relations are, however, purely qualitative, and alone are entirely insufficient to establish any deep-seated connection between the action of the finely divided metals on the one hand, and organic ferments on the other.

The first investigation was a quantitative study of the action of the colloidal solutions of the metals as compared with the action of organic ferments. As this has already been reviewed, a brief reference to the conclusions reached by Bredig and von Berneck is sufficient.

It was shown in the first place that very small amounts of metal can effect the decomposition of enormous quantities of hydrogen dioxide, just as very small amounts of organic ferments can effect relatively large transformations. This analogy is, however, not necessarily very striking.

It was then shown that the finely divided metals do not enter into the reaction which they effect. The method by which this was proved was considered at sufficient length in the earlier review.[3]

Substances which behave in this manner, *i. e.*, act by means of their surface alone without entering into the reaction, are known as *Catalyzers* and such reactions are termed *catalytic*. The collodial solutions of the metals, then, act catalytically. It is also well known that enzymes act catalytically. They do not enter into the reaction as such, but act simply by contact through their surface.

This analogy between the action of the colloidal metals and the enzymes is much closer than that first referred to, and makes it probable that the relations between the two are really deep-seated. There is, however, a relation far more striking than either of those considered above.

It is well known that the enzymes are very sensitive to certain substances, which, in infinitesimal quantities, may retard or entirely prevent their action. Thus, hydrocyanic acid, carbon bisulphide, hydrogen sulphide, and the like, when present in mere traces, may interfere seriously with the action of certain ferments. It has been shown that the merest trace of substances like the above may seriously interfere with the action of the colloidal solutions of the metals—may *poison* the finely divided metals. It is with this subject that the present review has to deal directly.

In their first paper certain qualitative observations were made by Bredig and von Berneck on the poisonous action of a few substances on colloidal solutions of platinum. This subject has subsequently been studied quantitatively by Bredig and Ikeda.[4] They have worked with colloidal solutions of platinum, and have studied the rate at which these decompose hydrogen dioxide. Solutions of certain poisons of different concentrations were added to the colloidal solution of platinum, and the rate determined at which the colloidal solution, after it had been poisoned, decomposed hydrogen dioxide.

The velocity of the decomposition was found to diminish when the poison had been added to the colloidal solution, and the amount of diminution depended upon the amount of poison present. The diminution in the velocity was worked out numerically, but the results can be seen most readily by means of curves.

Fig. 1 represents the results obtained with hydrocyanic acid as the poison. The abscissa represents the time during which the reaction has proceeded. The ordinate, log

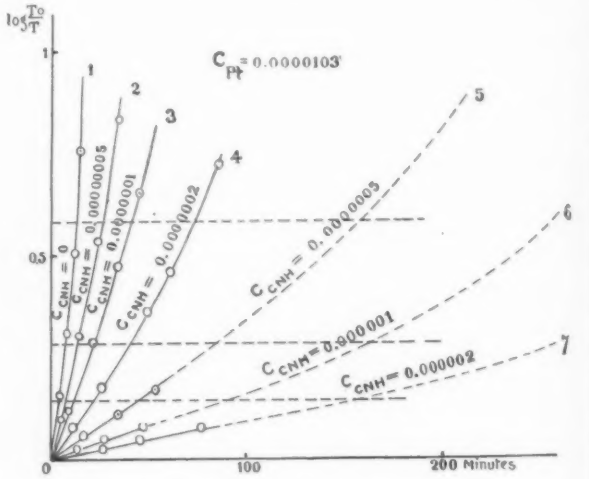


FIG. 1.—HYDROCYANIC ACID.

$\frac{T_o}{T}$, is really log $\frac{A}{A-x}$, in which A is the amount of the hydrogen dioxide originally present and x the amount decomposed. Without going more fully into this expression it may be stated that it is obtained by integrating the equation for a first order reaction derived from the law of mass action.

The concentration of the colloidal solution of platinum which was used, C_{Pt}, contained 0.0000103 of a gram-atomic weight per litre. The concentration of the hydrocyanic acid which was employed, C_{CNH}, expresses the number of gram-molecules of the acid in a litre of the solution.

Curve 1 represents the reaction when no poison is present. Curves 2 to 7 represent the reactions when increasing amounts of the poison were present. Take curve 5. Here the reaction velocity has been reduced to about one-half, and this has been effected by a solution of hydrocyanic acid containing *one gram-molecular weight in two million*

[2] Ztschr. phys. Chem. 31, 258, (1899.)

[3] This BULLETIN, 114, 225, (1900.)

[4] Ztschr. phys. Chem. 37, 1, (1901.)

litres. Such a solution contains in a litre only 0.014 milligrams of hydrocyanic acid, and yet is capable of diminishing the action of the colloidal platinum to one-half.

Curves 4, 3 and 2 represent decreasing amounts of the poison as expressed by the values of C. Curve 2 represents the effect of a solution of hydrocyanic acid so dilute that it contains *one gram-molecular weight in twenty million litres* of the solution; and at such inconceivable dilutions the action of the platinum is considerably lessened.

It has been found that the action of the enzymes in the blood in decomposing hydrogen dioxide is very appreciably lessened by the addition of a solution of hydrocyanic acid containing 0.002 milligrams in a litre.

A careful examination of the curves brings out another very interesting relation. The curves for the most dilute

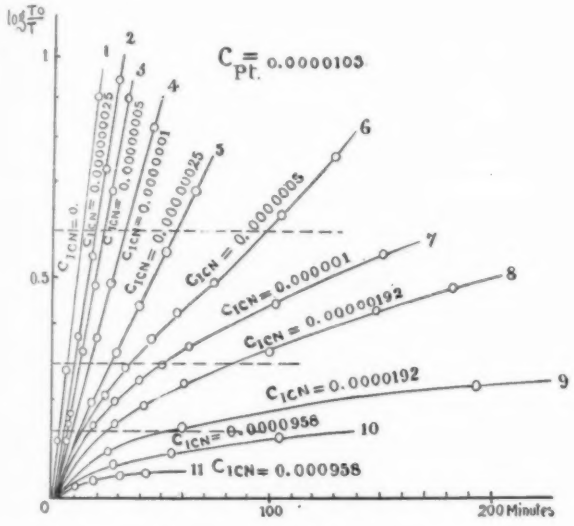


FIG. 2.—CYANOGEN IODIDE.

solutions of hydrocyanic acid are convex to the abscissa from the very origin. This means that the poison is having less and less effect as the time increases—the colloidal solution of the platinum is *recovering from the poison.*

Where the solution of the hydrocyanic acid is stronger (curves 5, 6 and 7) the curve is at first concave to the abscissa, and becomes convex only after a much longer time has elapsed. While the curve is concave to the abscissa the action of the poison is increasing, and the platinum begins to recover from the effect of the poison only after a much longer interval of time.

Similarly, it has been shown that the enzymes in the blood and ferments can recover from the poisonous action of minute traces of hydrocyanic acid.

It is pointed out that both of these effects are probably due to the oxidation of the hydrocyanic acid in the solution.

A more violent poison than hydrocyanic acid is cyanogen iodide. The results obtained with this substance are shown in Fig. 2.

Curve 1 represents the reaction when none of the poison is present. Curves 2 to 11 represent the results with increasing amounts of the poison. When the concentration of the poison is such that a *gram-molecular weight is contained in forty million litres* of the solution, the action of the platinum is very appreciably diminished. The diminution in the velocity of the reaction is increased as the concentration of the poison becomes greater and greater. When the poison has acquired a concentration of a gram-molecular weight in about thirteen million litres, the velocity of the reaction is reduced to one-half.

These curves show that the colloidal solution of platinum can *recover* also from the poisonous action of cyanogen iodide, provided the poison is not too concentrated. If the dilution is greater than a gram-molecular weight in a million litres (curves 2 to 6) the platinum can recover in part from the effect of the poison, as is shown by the curves becoming convex to the abscissa. If the concentration is greater than one million litres the curves remain concave to

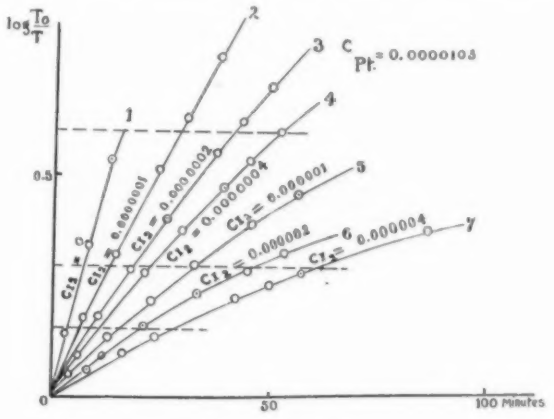


FIG. 3.—IODINE.

the abscissa, which means that the colloidal solution cannot recover from the effect of the poison.

It should be noted that cyanogen iodide is one of the strongest poisons toward blood and protoplasm in general.

The effect of one other poison will be taken up, since it represents the case where the colloidal solution cannot recover at all from the poisonous effects. This poison is *iodine.* The results obtained are shown in Fig. 3. From what has already been said concerning the curves in Figs. 1 and 2, the curves in Fig. 3 are self-explanatory. It will be observed that none of the curves become convex to the abscissa, which means that no matter how dilute the poison, provided that it is sufficiently concentrated to have any appreciable effect, the platinum cannot recover from its influence.

The curves show also that iodine is a powerful poison on the colloidal metal, and it is known to be an intense blood poison.

In a manner exactly analogous to that described above, Bredig and Ikeda have studied the poisonous action of about thirty substances on colloidal solutions of platinum.

The strongest poisons in addition to those described above are mercuric chloride, hydrogen sulphide, sodium thiosulphate, carbon monoxide, phosphorus, phosphene, arsene, mercuric cyanide and carbon bisulphide. From the effect of some of these substances the platinum was able to recover, as in the cases of hydrocyanic acid and cyanogen iodide, while the action of others remained permanent as with iodine. Among the substances which are less poisonous to the platinum are aniline, hydroxylamine, bromine, hydrochloric acid, oxalic acid, amylnitrite, arsenious acid, sodium sulphite, and ammonium chloride.

The weak poisons are; phosphorous acid, sodium nitrite, nitrous acid, pyrogallol, nitrobenzene, ammonium fluoride, etc.

A few substances were found to be nearly neutral in their action, such as potassium chlorate, ethyl alcohol, amyl alcohol, ether, glycerol, oil of turpentine, and chloroform; while formic acid, hydrazine, and dilute nitric acid increased the activity of the colloidal platinum in breaking down hydrogen dioxide.

At the close of their paper Bredig and Ikeda compared the action of a number of substances on the power of colloidal platinum on the one hand, and of blood on the other, to break down hydrogen dioxide. Their results are given in the following table:

RETARDATION OF THE CATALYTIC DECOMPOSITION OF H_2O_2.

	BY BLOOD.	BY COLLOIDAL PLATINUM.
Hydrogen Sulphide	No catalysis.	Catalysis very strongly retarded.
Iodine	Catalysis very strongly retarded.	Catalysis very strongly retarded.
Mercuric Chloride	Catalysis almost entirely prevented.	Catalysis very strongly retarded.
Mercuric Cyanide	Catalysis very considerably weakened.	Catalysis very strongly retarded.
Hydrocyanic Acid	Catalysis very considerably weakened.	Catalysis very strongly retarded.
Hydroxylamine	Catalysis entirely prevented.	Catalysis strongly retarded.
Amyl Nitrate	Catalysis strongly retarded.	Catalysis strongly retarded.
Nitrobenzene	Catalysis appreciably weakened.	Catalysis slightly weakened.
Pyrogallol	Catalysis not lessened.	Catalysis slightly lessened.
Arsenious Acid	Catalysis inappreciably lessened.	Catalysis slightly lessened.
Potassium Chlorate	Catalysis slightly decreased.	Catalysis not decreased.
Aniline	Catalysis strongly increased.	Catalysis considerably decreased.

That there is a general agreement between the action of poisons on organic and inorganic ferments is unmistakable from the above results. Certain differences, however, manifest themselves as in the case of aniline, and it is impossible to say at present what these mean.

An impartial examination, however, of all the evidence bearing upon the relation between organic and inorganic ferments will certainly lead to the conclusion that these analogies are not accidental. The state of mind of Bredig on this whole problem can be seen best by quoting the following paragraphs from his monograph on this subject:

"All these facts point to an unmistakable analogy between the contact actions in the inorganic world, and the actions of ferments in the organic world. As in the case of my colloidal catalyzers, we are dealing with reactions in which enormously developed surfaces are involved, so is it probable that the same condition obtains in the actions of ferments, enzymes, blood corpuscles, and oxidizing and catalyzing organic substances. We see, therefore, that the organism develops its enormous surfaces in the tissues and colloidal ferments not only because it requires osmotic processes, but on account of the very great catalytic activity of such surfaces. If, as Boltzmann says, the war for existence which living matter must wage is a war about free energy, certainly, of all the forms of free energy the *free energy of surface* is the most important for the organism.

"In conclusion, I need scarcely state that I do not maintain that there is any mysterious identity between the metals and the enzymes. But without exaggerating the overwhelmingly large number of analogies, we are compelled to regard the colloidal solutions of the metals, in many relations at least, *as inorganic models of the organic enzymes.*"

The author would refer the action of the colloidal solutions of the metals, the enzymes, and catalysis generally to the *surface energy* possessed by these substances. We are thus led one step nearer to the solution of the nature of catalysis, which underlies so many of the processes of living as well as dead matter.

GREEK IN MEDICINE.[1]

By Achilles Rose, M. D., New York.

The subject which I have the honor to place before you is as important as it is fascinating. Eloquence I have none; fortunately for me no eloquence is required to excite your interest in the cause I am pleading.

The significance of Greek in Medicine can only be appreciated when we know the complete history, or at least certain facts of the complete history, of the Greek language. Our schools give us only a fragment of this history, because our schoolmasters, our college professors, know only a fragment of the Greek language; of its history they know still less.

[1] Read before the Book and Journal Club, Baltimore, Feb. 19, 1902.

The history of ancient Greek literature even is not the history of ancient Greek language, for that part of the ancient Greek literature which has been transmitted to us through the classical writers is an artistic product beginning with Homer and ending with the end of paganism. This artistic product was not the language really spoken.

Ancient Greek language begins with the origin of the Greek race and extends to the present day.

Greek is the oldest of all the living languages of civilized nations. From the classical period of the Attic orators down to the present day it forms an unbroken continuation of classical Attic.

We shall see how Byzantine and Mediæval Greek, or Middle Greek as it is called, thus far have received but little attention; they have, as a rule, not met with any sympathetic interest on the part of the classical students. These phases of the language have on the contrary been branded with unmerited reproach and scorn, and the Greek spoken at the present time is made the object of ridicule and discredit, even by men otherwise of learning, who have no idea of living Greek and who repeat only what they know from hearsay, or what they have read in papers and periodicals written in an inimical spirit towards Greece.

In the middle of the fifteenth century, after the fall of Constantinople, Greek fugitives came to all parts of Europe; and it was they who introduced the study of Greek and laid the foundation of the "Western" school. The first act of this school, still in its infancy, was to do away with the traditional pronunciation and then to declare Greek a dead language.

They cut off all direct connection between the verbal and the written tradition of Greek. At this time the study of philology in regard to Greek became unscientific and has remained so ever since, for the study of a language like the Greek must not confine itself to written tradition. Only the study of the two forms of tradition in connection with each other renders possible a scientific understanding of the old phase as well as of the new; the two forms of tradition influence each other. One of the influences is that of the older monuments of literature upon the later phases of the language, and this influence is the greater the more the old literature is manifold, and the more diligently it is studied by the descendants.

With the close of the 6th century B. C. the dialect of the Athenians, the so-called Attic, prevailed over all other sister dialects, and eventually absorbed them. It was the Attic, because Athens, particularly after the Persian wars, rose to absolute dominion over all other Greek communities, and finally became the metropolis of all Greek races.

There has never existed a language which permitted greater distinction of expression or style than the Attic. To speak and to write Attic was, on account of its richness, as easy as it was difficult to do so with elegance. In speaking of Attic, therefore, we must not infer that all Athenians and Atticized Greeks wrote and spoke the classical Attic portrayed in the classical literature. We have to take into account, as is shown by force of logic, by historical investigation, by modern analogies and by daily experience, that it is an indisputable fact that no writer uses the same diction, both in writing and in every-day speaking. Every Greek at present, for instance, is at liberty to use the colloquial language or even his own dialect in every-day intercourse; but when he proceeds to commit his thoughts to paper he is compelled to follow the Attic grammar—the style, as a matter of course, varies with the degree of individual education of the writer. The colloquial speech, therefore, is hardly presented in literature. It never attained recognition or favor among the educated writers; on the contrary, there has been at all times a general prejudice against it, and the writers of all times have made it a special point of honor and pride to make the least possible concessions to the popular vocabulary. It is true that, during the Turkish rule, attempts were made to use the vulgar language in literature. Roman Catholic priests, for propagandist purposes among the Greeks, translated the liturgy into the idiom of the people. The Greeks are especially careful not to employ common language when they speak of sacred things. For these, and many more reasons, the Catholic priests did not succeed. To what extent the whole Greek nation is opposed to the idea of introducing popular language into literature, has been demonstrated again quite recently by the events in Athens which took place when the Queen attempted to introduce a translation of the gospel into vulgar Greek. Here, in New York at least, there was a most unanimous expression of horror among my Greek friends in regard to the idea of this translation. Reading the Greek criticisms in the papers of the time when the first specimens of this translation were published I learned how rich the Greek language is in derisive adjectives, for the papers made use of the entire wealth of these adjectives against the translation.

These facts show how unjust it is to draw a parallel between classical Greek, in its artistic as well as its artificial form, and the everyday colloquial Greek of the masses of the present day; but such injustice is the rule, even among philologists.

The final subjection of Greece to the Macedonian rule ended her glorious days. At that time, the conquests of Alexander the Great, in Asia and Egypt, threw the East open to the Greek population. Multitudes of Greeks came to Asia, Egypt, North Africa, Southern Europe. The Greek language, then already Atticized, was triumphant everywhere, became an international language. The great mass of Hellenes, who had come with Alexander the Great, were forced more and more, in order to understand each other, to give up their provincial idioms, and to adopt the Attic dialect. This generalization of the Attic was called the κοινή, that is, the general language. Attic became κοινή, and this is identical with New Greek. Let us bear this fact in mind, because it is of paramount importance. New Greek originates from the κοινή, not from the dialects. Attic κάτνή has conquered the old dialects. The inscriptions of the time

of Alexander the Great at first were partly in dialect but finally nothing dialectical remained.

There were some writers during the 1st and 2nd centuries of the Christian era, who for love of archaism, tried to write the old dialects; but they found themselves unable to do so, and committed all sorts of errors in their writings. This would have been impossible if the old dialectical forms had still been alive in their old purity, that is, if they had been spoken still.

Then the balance of culture and learning shifted to the East, Alexandria becoming the principal centre of classical education and learning; hence this period is called the Alexandrian.

The Greek population had settled abroad among alien races as soldiers, colonists, tradesmen and the like, and formed only a minority among the natives and a sort of colonial—the Levantine—Greek developed, but whatever foreign elements had been introduced at that time were eliminated again in subsequent periods.

During the Græco-Roman period (150 B. C. to 300 A. D.) the Romans, instead of Romanizing their Greek subjects were Hellenized by them. Whatever Latin words, referring to Roman associations and novelties had been adopted in a more or less Hellenic form during this period, were also eliminated again.

While thus the language was in danger of adopting foreign elements, in danger of comparing unfavorably with the Attic of the glorious olden times, many scholars at that time, and later on the great majority of their successors, endeavored to check the progress of this "common" tongue, as they called it, that is, the unclassical Greek, and to revive the pure Attic. They were called the Atticists or Purists. The extreme Atticists thought one should not only imitate the old genius, the right composition of speech, the pure and simple diction of the ancients, as was the teaching, for example, of Dionysios of Halicarnassos, but also to copy scrupulously the words and forms.

The literature of the extreme Atticists or at least the writings of these Atticists gives no picture of the language of their time.

The writers of the Graeco-Roman period were 1. Atticists; 2. Conventional writers who wrote Attic with many concessions to the spirit of the time; 3. The Levantine Group, represented by the Asiatic Greeks and Hellenized foreigners. The New Testament is written in Levantine Greek.

Christianity originated in Asia Minor, which was ruled by Rome but spoke Greek. Christianity sprang up in the midst of the masses, borrowed from Hellenism its language, and became a part of the Hellenic race and nature. The very founder of Christianity (presumably) and his apostles (certainly) preached and wrote in Greek. Nothing has been more potent in the preservation of the old Greek than the influence of the Church.

When the Roman Empire was dying out it had the good fortune to be absorbed in the life of Greece and it derived from that union a renewed energy which secured for it another millenium of existence in the Byzantine Empire. The first Byzantine emperor, Constantine the Great, adopted the Christian faith and was the first Christian emperor.

Until recently the Byzantine era was the least known and the most obscure in the field of historical study, the history of the Byzantines, of their Greek and Christian state over a thousand years of existence, was treated with great injustice, exaggerated severity, and contempt. Two writers especially, Montesquieu and Gibbon, have done a great deal to promulgate error and injustice in regard to Byzantine history. There exists even at this present day no fair history of the Byzantines in the English language. What applies to Byzantine history applies likewise to Byzantine literature and language. The only collections of importance from this literature were made in France during the 17th century under the auspices of Louis XIV, in the Corpus Historiæ Byzantinæ, a work of 42 very large volumes in folio; and now the works of the Byzantine medical writers are collected and published under the editorship of Foustanos, the editor of the Greek Medical journal Ἰατρικὴ Ἡρόδοτος of Syra.

Philologists are accustomed to distinguish ancient Greek from modern Greek without considering middle Greek, notwithstanding the fact that there is nothing in modern Greek which has not come from ancient Greek by way of middle Greek. The study of this latter phase of the language furnishes the best information for the scientific knowledge of modern as well as of ancient Greek.

Ten years ago there appeared in the German language a work, modestly called "Einleitung in die neugriechische Grammatik," by G. N. Hatzidakis, a Cretan, Professor of Philology at the University of Athens, and this book gives more valuable instruction on the history of Greek than all the other books, written on this subject, together.

A great number of forms, words, and constructions, which, according to the literature of several ages, one would believe to be lost, can be found to-day in the different dialects spoken in different parts of Greece. Phenomena which would appear to be modern can be shown through these dialects to have originated in the oldest times; indeed many old phenomena have been preserved to this day in the dialects, although the literature of the middle ages and of modern times bear no witness to them. In view of these facts, we can readily see that only a man like Hatzidakis, a philologist, a native Greek, who can identify himself with the Greek peoples' dialects is qualified to give the history of Greek.

During the Mediæval period (from 1000 to 1450) in Greece proper, a vocabulary of the feudal reign of the Franks, chiefly bearing on chivalry and gallantry was mixed with Greek, but never struck root in the Greek language. All these foreign words soon passed into oblivion. Similar considerations apply to the succeeding dominion of the Venetians, whose institutions never became popular in Greece.

A striking illustration of the failure which attended the efforts of Venice—the most civilized and mighty of all foreign rulers in Greece—to assimilate her Greek subjects, may be found in the present state of Crete. This island remained

under the Venetian dominion, and formed a dependency of powerful organization for an unbroken series of more than 450 years (1210 to 1669), yet with all that, hardly any native Cretan Roman Catholic is to be found on the island, and the Cretan vocabulary of to-day does not preserve fifty words which can be traced directly back to the Venetian domination.

It is hardly necessary to speak at this present time of the witty arguments of Fallmerayer; they have no foundation, and have been discredited long ago by all serious investigators. The fact is that, except a very few names of hills and rivulets, there exists not a single Slav word in the Greek language spoken and written to-day.

The Greek of to-day as taught in the schools throughout Greece, the official language of the government of Greece is pure Attic Greek, as pure as it ever was. It is the immortal Greek in all its youth and beauty, free from foreign elements.

Our Græco-Latin onomatology is evidence that our science originated in Græco-Roman antiquity and these classical languages in our onomatothesia have given our science an international character and established a union of the different peoples for the good of science.

Our Western civilization has adopted from the Greek literature the really moving thoughts and the facile forms. The school of Athens formed the background of all learned activity. Homer, Aristotle and Plato have continued to be, up to our time, the teachers of mankind. The classical writers of Rome were in their works a long way behind the Greek authors; indeed, the best among them are indebted to their Greek antecedents for their education. The Latin language was the first one which borrowed from the Greek, and the languages of all civilized nations, above all the German, French and English, have done likewise. The number of Latin medical terms is inferior to the number of words from the Greek, and medical writers, when they coin new words, prefer to borrow from the Greek.

Greek has been the international language of the world, and Virchow, in his inaugural address as rector of the University of Berlin, said that it was from the beginning a weak side of the humanistic educational institutions to favor Latin as the international language of scholars.

But we will not dwell to-night on Latin, nor shall I speak of Greek as the international language for scholars. I shall confine myself to speaking of Greek in medicine, and to this end I may be permitted to say first a few words on our onomatology in general.

Twenty years ago, in an article which appeared in the Berliner Klinische Wochenschrift, Virchow said: "Is it not justifiable at the present time to remind the physicians that they should use all possible exertion to show their scientific training in all, even in apparently small things? Is it not the first characteristic of a man of science that he should understand how to employ the language of science? The first thing by which the expert demonstrates his qualification is the correct use of technical terms; this specially causes respect for him, and nothing more enhances his utility"; and he closes his remarks with the wish that his views might be generally adopted.

If we address this admonition to the medical profession at large we shall have to say: Would it not be justifiable at the present time to remind the profession to prove our science by establishing a scientific onomatology?

The incorrectness of many of our medical terms cannot be denied, but the extent of this incorrectness is larger than most of us have ever conceived; the evil is increasing every day, and the remedies thus far suggested have only brought confusion; they are not, and have not been, taken seriously; they have only brought discredit.

Hyrtl, who was without doubt the best classical scholar among the medical men of his time, gave us a work entitled Onomatologia Anatomica, in which he exposed a great number of terms as being incorrect, ridiculous, absurd, and, hence, unscientific; but he says quite modestly: "The idea of reforming the anatomical language could not enter my mind. To this end is required a committee of anatomists, selected *ad hoc*, who are at the same time linguists, and as cöoperators, philologists, an Academia della Crusca Anatomica. I merely had the intention of demonstrating to those who will take the pains to read this book the necessity of reform."

It may appear audacious and impious on my part when I say: with all his classical training, Hyrtl himself added errors, because in one requirement he was wanting, like the rest of the would-be reformers, he had no idea of living Greek, for he quotes colloquial or vulgar Greek and calls it New Greek.

Henle, an anatomist of the same high rank as Hyrtl, attempted to simplify the anatomical onomatothesia, but only added a new complication, because his suggestions were not uniformly accepted; he likewise did not consider the existence of living Greek.

Virchow, in his inaugural address on Greek in medicine, had in view only Greek as taught in our schools; living Greek he did not mention. All the lexicographers outside of Greece have been obliged to ignore it, because the medical word-makers of our times have ignored it, or have quoted vulgar Greek, which our Greek colleagues exclude from scientific literature, or, worst of all, they have introduced words which they stigmatized as "New Greek" which are not Greek at all.

It is not necessary to enumerate all the vain attempts at reformations of our onomatology; a single one, and I select the one which is most conspicuous at present, will suffice. We shall see why they were all of no avail, but we shall also see how we really can secure scientific onomatology which will stand as long as our science is taught, and which will be beyond all criticism. But let us first speak of the nomenclature of the German Anatomical Society.

The German Anatomical Society appointed a committee and raised the necessary funds, about three thousand dollars, from contributions by different medical academies to furnish an onomatology with all names in grammatically correct

Latin. *All Names in Latin!* The members of the committee were all prominent German anatomists and all thorough classical scholars; they worked hard for seven years; thus they conducted an immense amount of correspondence and had many meetings in Munich, Vienna, Goettingen, Strasburg and Basel; their sessions in Munich, for instance, commenced at 8 A. M. and lasted until 6 P. M., in Vienna even until late into the night. In the year 1895 the new anatomical nomenclature—Nomina Anatomica—as the title is, was published, and whoever chooses may accept the names of this onomatothesia. Most of these names are by no means Latin, but Latinized Greek, or they are unscientific hybrids. In some of them of more than two syllables we find the syllables alternately taken from one and the other language.

The German Anatomical Society has undertaken a thing which is an impossiblity—namely, to restore to life, to develop further, a dead language.

Had the society, however, taken the living Greek for a basis instead of the dead Latin, had they consulted real Greeks, the professors of anatomy of the University of Athens, they could have fulfilled their promises, executed all their intentions without the arduous labor of seven years.

The result of the labors of the German Anatomical Society demonstrates that even an academia, as suggested by Hyrtl, will not succeed so long as it applies dead language to new conceptions, and treats a living language as it were a dead one.

As mentioned before, the classical languages in our onomatology give our science an international character and continue to establish and secure more and more a union of the different nations for the good of science. But science requires that our onomatology should be correct and scientific, and this it can only be when new conceptions are named in Greek; and no new formation in Greek can be proven correct that does not meet with approval of our Greek colleagues.

There exist words in our everyday language which a Greek friend of mine is in the habit of calling ἑλληνοφανής, that is "Greek seeming or supposed Greek," as for instance the word bicycle or telegram; they are an impossibility in Greek and such ἑλληνοφανῆς words we have in great number in our onomatology. Most of them are simply horrid, notwithstanding that they were coined by men of great learning, by profound classical scholars. Every one of us has had ample opportunity to observe how awkwardly English is spoken and written by those who have learned the language in some German or French schools by means of grammar and lexicon only, and this awkwardness is manifested by our medical word-makers who coin what they suppose to be Greek terms without being familiar with esoteric Greek, as only those who are Greeks or who have lived among them can be. Now let us see how the Greeks go to work when new formations are required: When Greece had regained her liberty after almost four centuries of Turkish bondage, a regular government was to be erected. Countless numbers of demands were made on the language. A new life, a culture of which there had been no idea before, appeared suddenly before the Greeks. The language had to keep pace with the many new political, scientific, technical, commercial, journalistic requirements. Another nation would certainly, under such circumstances, simply have adopted with the foreign ideas the words also of foreign people, and would have formed a hybrid language. Not so with the Greeks. Their history, their national pride led them to exclude foreign words, led them to take the necessary elements from the old Greek to create new symbols for new ideas. Constructions and forms were remodelled after the old Greek, incorrect elements, when discovered, were extirpated with more and more severity and tact, and so it is to-day. Before a new formation is introduced into the regular language it has to stand a severe test and criticism. Nothing will be accepted and introduced into the regular language which deviates in any way from the genius of the Attic language. We need not, therefore, fear that terms which we might accept from our Greek colleagues would be contrary to the spirit and the form of classical Attic.

The facts I have presented to the Book and Journal Club of the Medical and Chirurgical Faculty of Maryland will, I am certain, find an echo in the medical profession. There exists a well-pronounced demand by all true men of science for purity and correctness of medical language. Professor R. Kossmann, of the University of Berlin, whose name as a gynecologist is familiar to you, but who is not only a gynecologist, but has also distinguished himself as a philologist, says: "In forming our pathological and surgical technical terms we can by no means dispense with Greek, and so long as we admit that the retention and introduction of Greek synonyms for pathological and surgical expressions is a necessity, we have for the sake of correctness and beauty of language to decide to go a step further, to do away with hybrid terms and establish pure Greek synonyms in their place. Whoever has familiarized himself with classical literature will find it more and more intolerable to name a scientific conception by a Greek- or Latin-like sounding word monstrosity, especially when he knows the real Greek name."

Dr. H. Zimmerer, a distinguished professor of philology in Germany, one of the exceptions among the German philologists in so far that living Greek is familiar to him, honored me by the invitation to cöoperate in a new edition of a medical lexicon. In my turn I secured the assistance of a Greek friend, Basilios Leonardos, doctor of medicine and philology, director of the Museum of Inscriptions of Athens. I thus learned that professors of the University of Athens would be ready to aid in case a thorough revision of our onomatology in regard to Greek should be intended.

I shall congratulate myself if I have succeeded in demonstrating the possibility of a scientific onomatology and displayed the significance of Greek in Medicine in a new light.

ON SYPHILITIC DISEASE OF THE CEREBRAL ARTERIES.

BY F. C. GOLDSBOROUGH.

(From the Pathological Laboratory of the Johns Hopkins University and Hospital.)

The literature on the subject of the vascular changes in syphilis has been so frequently and so well reviewed that it need be merely briefly summarized here. More detailed references may be found in the papers of Abramow,[1] E. Meyer[2] and Oppenheim.[3]

Heubner[4] first accurately described these changes as specific in their characters and easily distinguishable from the ordinary arteriosclerotic processes. The specific nature of the changes has been doubted by many authors, but seems now to be pretty generally accepted. The definitely circumscribed thickenings of the wall leading often to obliteration of the vessel, the almost acute course, and the occurrence in young people often without general arterial affection, characterize the syphilitic changes fairly well. Instead of a degenerative lesion in the wall which leads to a diffuse widening of the vessel often with calcification, we have a nodular thickening which is definitely gummatous in its histological characters which narrows the vessel and in its late stages may undergo a caseous degeneration.

As to the point of origin of this gumma formation opinions differ widely. Heubner considered the thickening as primarily due to a proliferation of the endothelial cells with the formation of a mass of spindle cells and later affection of the outer coats of the artery.

Baumgarten[5] agreed with Heubner as to the specific nature of the process, but concluded that the changes began in the adventitial coat. There when they constituted a distinctly gummatous formation they could be recognized as syphilitic, while the much less advanced alterations in the corresponding intima were as yet not particularly specific in character. Köster[6] went even further in denying the occurrence of any proliferation of the endothelium. Recently Junzo Nagano[7] clearly states his conclusions which in general agree with those of Baumgarten, as follows: "In the first place only the adventitia is constantly diseased—where the intima is affected the adventitia is always involved, but the reverse is not true. I do not hesitate therefore to consider the adventitial change the primary and essential, while the changes in the intima are secondary. While the intima is greatly thickened in some places, still in many this change is quite lacking. The media, on account of its very slight alteration, does not enter into consideration as a point of origin for the process."

As to the part played by the media, Rumpf[8] put forward the idea that the syphilitic process originated from the capillary system of that layer and thence extended to the adventitia or to the intima. Finally Oppenheim declared that the three coats of the artery may become diseased independently of one another.

Thus we find these authors expressing every possible opinion as to the point of origin of this syphilitic new formation in the vessel wall. Certainly in advanced cases all of the walls are found involved, and evidence as to this point can only be expected from examination of the early stages in the process or possibly examination of the advancing margins of the diseased portions of the vessels.

The following case occurred recently at the Johns Hopkins Hospital:

Mary D., colored, aged 30 years, admitted March 22nd, 1901, complaining of headache. There was no history of gonorrhea or lues. She had had rheumatism 2 years ago lasting over 5 months. Had given birth to two children, one 10 years ago and one 9 years ago; the first was still-born, the second died, aged one and a-half years.

She complained of having had headache for more than a year and five months; at the onset there were several attacks of fainting lasting through a week at a time. Recently her eyes had failed and she had become stupid and slow mentally. There were no convulsions.

On admission there was a bulging of left eye; no paralysis, although there was some weakening of the muscles of the right side of face and of left arm. Reflexes were normal.

Physical examination of the abdominal and thoracic viscera negative. The blood count at this time showed:

Hæmoglobin	40%
R. B. C.....	2,700,000
Leucocytes	8,000

On April 22nd definite optic atrophy developed. On April 29th lumbar puncture was performed with no results. On May 10th there was ptosis of the right lid; reflexes became exaggerated; there was stiffness of the legs, arms and neck. May 30th patient went into coma and died June 2nd.

Urine showed traces of albumin, but no casts.

Autopsy was performed June 2nd.

The viscera in general showed no abnormality except for the presence of small caseous masses in the liver, which were surrounded by widely radiating scars; in other portions of the liver such scars occurred alone.

In exposing the brain and cord, the vessels of the pia over the surface of the brain were found to be much injected and

[1] Abramow, Ziegler's Beiträge zur Path. Anat., 1899, Bd. 26, S. 202.
[2] E. Meyer, Centralbl. f. Allg. Path. u. Path. Anat., 1898, Bd. IX.
[3] Oppenheim, Handb. d. Specielle Path. u. Therap., herausg. v. Nothnagel, 1896, Bd. IX.
[4] Heubner, Die luetische Erkrankung der Hirnarterien, Leipzig, 1879.
[5] Baumgarten, Virchow's Archiv, 1878, Bd. LXXIII, S. 90.
[6] Köster, Berliner Kl. Wochenschr., 1876, Nr. 31.
[7] Junzo Nagano, Virchow's Archiv, 1901, Bd. CLXIV, S. 355
[8] Rumpf, Die syphilitische Erkrankung des Nervensystems, Wiesbaden, 1887.

there were here and there minute hæmorrhages; these were also found on the under surface of the frontal lobe. There was a marked opacity of the pia along many sulci, especially in the frontal region and longitudinal fissure; this was also marked in the fissure of Sylvius. Many of the arteries in this region showed minute greyish nodules scattered along their course. This condition was especially marked on the basilar portion of the brain where, over the optic chiasm, there was an opaque mass forming a thick envelope which involved also the carotid artery; each of the cranial nerves was developed separately in such an opaque mass.

Over the pons and between the cerebellum and cerebrum there were similar irregular yellowish masses which in the peripheral portion were sometimes translucent. These formed at the junction of the vertebral arteries a mass measuring 1.5 cm. in diameter.

Over the dorsal surface of the cerebellum the beading of the arteries was especially well seen. The cerebral ventricles contained no excess of fluid and the ependymal surfaces were smooth.

On section the brain substance appeared normal throughout.

Microscopical Examination.—Sections of several parts showed that the brain was practically normal, although here and there an increase in the number of perigangliomic cells was observed. The lymph-sheaths of the small vessels were filled with deeply stained small mononuclear cells. Minute hæmorrhages were also to be seen about some of the smaller vessels. Some of these vessels showed a hyaline degeneration of their wall which was somewhat thickened, homogeneous in appearance and without nuclei.

Sections were made through the thickened portion of the meninges in various places, and also so as to include the nodular thickenings of the vessels. The smallest of these were preserved for investigation and sections, both transversely and longitudinally, were made.

The meninges in the neighborhood of the affected vessels proved to be somewhat thickened, and for the most part infiltrated with cells; the greater number were deeply stained, small mononuclear cells with very scant cytoplasm; polynuclear cells, however, occurred here and there.

In a cross section of one of the small thickened vessels the lumen was found to be disproportionately small, but filled with blood and lined with intact but somewhat thickened endothelium. Outside of this there was found a mass of epithelioid cells in a dense reticulum. These cells were variable in size and their form was sometimes much elongated. This tissue was somewhat eccentrically arranged so that on one side of the lumen it formed a thick mass where the cells became less numerous and the intercellular substance increased; here much nuclear distortion and fragmentation occurred; there was, however, no actual caseation. The whole mass was surrounded by the much thickened elastic lamella which was very tortuous on the side near the lumen, but stretched out smooth over the thickened mass on the other side.

The media was intact over the part of the vessels where the thickening was least; the rest of the media was stretched out thin and its laminæ were spread apart by the invading cells which were themselves undergoing fragmentation. The nuclei were much misshapen and were apparently disintegrating.

In the adventitia the tissue changes were especially marked. There there was great proliferation of the spindle-shaped connective tissue cells or epithelioid cells which made up the bulk of a considerable mass of tissue. Among these were seen many small round cells. Giant cells were infrequent, but occurred sometimes. They were more often seen just inside the elastic lamella. Very characteristic, however, was the great distortion of the nuclei of the epithelioid cells in areas in the nodule surrounding the patches of caseation which pretty regularly appear after the nodule had reached a certain size. The extension of such a caseated area toward the media led to the peculiar disintegration observed in the stretched-out portion of that layer.

In several longitudinal sections of such a vessel the relations are even clearer. The spindle cells of the subendothelial tissue are arranged almost entirely longitudinally, so that that tissue appears like a well-defined fibrous tissue. This thickening of the intima seems to correspond generally with the areas of thickening of the adventitia, but it is important to note that practically always the adventitial changes were obviously more advanced than those of the intima, and it was there that caseation first appeared in each case.

The condition was practically the same except in degree in all the vessels examined. The only change in some of the very small ones was a diffuse infiltration of the adventitia with lymphoid cells.

In some places more than one endothelium-lined lumen was seen within the tissue inside the elastic lamella. In the explanation of this phenomenon, Rumpf's idea that widened newly formed vessels in the tissue which goes to obliterate the original lumen give rise to these channels, seems more plausible than their interpretation as parts of the original lumen which have been partitioned off. There seemed to be a new formation of muscle about the endothelium of these secondarily formed blood channels. The membrana elastica in the affected vessels is generally enormously thickened, but no definite splitting or reproduction of the membrane has been observed.

Certain portions of the central nervous system itself were involved in these processes; the optic tract, for example, showed on section a dense infiltration with small round cells which infiltrated along the septa dividing the bundles of nerve fibres. The infiltrating mass was found to be continuous with that surrounding the carotid artery, although the carotid artery internally showed only the changes ordinarily seen in arteriosclerosis.

Also in the pons underlying the caseous masses about the basilar artery, there was a deep involvement of the nervous tissue which was much infiltrated and degenerated.

The ventral root of the 3rd cervical nerve was also extensively infiltrated with new tissue elements which pushed aside the nerve fibres and tended to destroy them. Some portions of this mass had even undergone caseation. A great many sections were stained for tubercle bacilli, but always with negative results.

From all this it is evident that we have in this case preeminently an affection of the vessels, the meninges and central nervous substance being relatively little involved. The lesions consist in thickening in the walls of the vessels which are histologically gummata with the characters recognized in gummata elsewhere. These gummatous lesions are most advanced in the adventitia, being generally accompanied by proliferation of the intima in a corresponding locality. The process, therefore, probably begins in the adventitia. It is, on account of its characteristic histological appearance, quite as specific in its nature as the tuberculous change in the vessels in tuberculous meningitis and is not to be confused with the ordinary arteriosclerosis.

In closing, I wish to express my appreciation and thanks to Dr. W. G. MacCallum, under whose supervision this work was done, for his very kind assistance and advice.

PARA-COLON BACILLUS.[1]

BY RICHARD P. STRONG, M. D., U. S. A.

A single case of infection with para-colon bacillus has come to us. This case was not seen during life, but the body was sent from Santa Cruz to Manila, where we observed it at necropsy. The following brief clinical history was obtained later from his hospital record at Santa Cruz:

"On July 5th, 1900, the soldier developed temperature. There were rectal discharges of blood and mucus. The chart shows the course of the fever to the time of death, July 31st. No departure from the ordinary type of typhoid fever."

The necropsy was held forty-two hours post mortem. The body was extremely emaciated; there was considerable post mortem change and gas in the sub-cutaneous tissues. The heart muscle was pale and yellow in color. The valves were all normal. The arch of the aorta and coronary arteries smooth. The lungs were also normal. The mesenteric lymphatics were swollen and some, along the small intestine, hemorrhagic. The spleen was very large and soft, and the pulp somewhat dark and considerably increased in amount. The liver showed advanced fatty degeneration. The capsules of the kidneys were not adherent; the organs were pale and yellow in color. Both the large and the small intestine were normal throughout, except for a moderate catarrh and a few superficial hemorrhages. The solitary and agminated follicles showed no lesions. The other organs were normal. Stained cover-slips from the liver showed a large capsulated bacillus, which, as it did not appear in aerobic cultures from the spleen, we regarded as probably bacillus aerogenes capsulatus; the cover-slips also showed smaller bacilli. Fresh smears from the spleen showed a few crescentic æstivo-autumnal malarial parasites, and a fair amount of malarial pigment. No hyaline bodies could be found.

As we desired a fresh typhoid culture, an extra number of cultures (four plates) was made from the spleen. After twenty-four hours a good number of colonies developed on these plates, which seemed similar to one another and resembled colonies of bacillus typhosus or delicate colon colonies. A number of these colonies were planted on all media with the following results:

On agar slants there was a moderately heavy growth; bouillon was at first clouded, after a time a sediment formed at the bottom and often a pellicle on top; hanging drop showed a motile bacillus; stained cover-slips decolorized by Gram; in glucose agar there was a moderate gas production; in lactose agar there was no gas production; saccharose was moderately fermented; litmus milk was at first reddened after fourteen to twenty-four hours, and then turned blue after about forty-eight hours; in Smith's sugar-free bouillon there was no indol production.

This organism was pathogenic for mice in doses of $\frac{1}{2}$ cc. The same bacillus was recovered from their blood and organs after death. Unfortunately no serum with the patient's blood was tried with the organism recovered.

As is well known, in 1897, Widal obtained from an abscess in the neck, about the œsophagus, after an attack of supposedly typhoid fever, an organism to which he gave the name para-colon bacillus. In the same group with this organism he placed bacillus psittacosis of Nocard and a bacillus of calf septicæmia of Thommasen. Widal's organism presented a strong agglutination for the blood of the patient from which it came.

In 1898, Gwyn reported a case of infection with the paracolon bacillus in a patient with all the clinical symptoms of typhoid fever. He isolated the organism from his patient's blood, whose serum agglutinated this organism, but not the bacillus typhosus. The organism differed in its cultural peculiarities from Widal's organism only in that it fermented saccharose.

Cushing has quite recently reported a third case of paracolon infection, the organism being obtained from an abscess of the rib following an attack of prolonged fever resembling

[1] From Report to Surgeon General "On Prevalent Diseases among troops in the Philippines." (Manila, P. I., Jan. 10, 1901.)

typhoid. The serum in his case also agglutinated the organism he isolated. Cushing worked with Gwyn's bacillus and concluded that neither his own nor that of Gwyn is capable of fermenting any other pure sugar than glucose. Our organism at present is still growing in the laboratory and a culture of it will be sent to you with this report.

In litmus milk with the organism, the change to the red color may be noticed in about fourteen hours; after about forty-eight hours the tube returns to about an equal shade to the control tube, or at the top it may be a little bluer. The bluing gradually continues at the top, while the litmus solution becomes gradually reduced and decolorized at the bottom. Cushing has stated that it can be readily understood if these organisms occupy, at one time or another, the alimentary tract during the course of fever associated with intestinal lesions, they might easily find their way into the portal circulation and cause a superimposed infection on the original disease. He also remarks, however, that the colon bacillus is rarely found beyond the limits of the abdominal cavity, while Gwyn's bacillus and his own were isolated, one from the peripheral circulation and the other from a surface lesion; also both gave an agglutination for the para-colon bacillus and not for the bacillus typhosus. Therefore he is inclined to believe that they were the specific cause of the disease which resembled, clinically, typhoid fever.

Unfortunately, we were not able with our case to throw much light on the subject, for no serum reaction was tried with the organism and the patient's blood from which it came. So, whether the organism was a post-mortem invader (as Reed has shown the bacillus icteroides, another member of the hog cholera group sometimes to be) or not, we cannot say definitely. At any rate, macroscopically, there were no definite lesions of the intestine to serve as the portal of entry for this organism if it did invade during life. However, no other cause for the death of the patient could be found. It is evident that he was suffering from some infection, as his temperature chart shows, and the few crescents and small amount of malarial pigment would not explain his death from malaria; besides, he had been treated with quinine.

We regret that this case is not complete, but trust that it will be remembered that the necropsy was an outside one, coming from a distance, that the body was already considerably decomposed, that we had heard only accidentally of its being a case of typhoid fever, and were anxious to obtain a fresh culture. For these reasons, also, no tissues were saved from the necropsy. However, it has been thought worth while to mention the case.

A CASE OF MULTIPLE PRIMARY ADENOCARCINOMA OF THE LIVER WITH CIRRHOSIS.

By Catherine H. Travis.

(From the Pathological Laboratory of the Johns Hopkins University and Hospital.)

Primary new growths in the liver have been the subject of a great deal of discussion in recent times. The literature has been collected by v. Heukelom,[1] and recently by Eggel.[2] The histogenesis of such tumors is made fairly clear by v. Heukelom.[1] Their relation to compensatory hypertrophy and to adenoma is discussed by Orth[3] and by Schmieden.[4] The coexistence of cirrhosis is also considered by these writers, but their conclusions agree very badly.

Although several kinds of malignant growth are described, one definite type stands out, in which are seen nodular tumor formations of atypical cells, an invasion of the vessels, metastases and cirrhosis.

A case of this type was put into my hands by Dr. Wm. G. MacCallum, whose kindly interest in the study and whose assistance I here gratefully acknowledge. I shall endeavor to give a description of the new growth and to discuss its histogenesis and relation to cirrhosis.

The case is that of T. S., a colored man, aged 40. With the exception of the ordinary diseases of childhood and an attack of influenza, this man enjoyed sound health up to the time of his last illness. He was, according to his own statement, free from venereal taint, and used alcoholics "only as a medicine."

In July, 1900, he began to have attacks of nausea lasting for several days. From this time he was troubled more or less by digestive disturbances. At Christmas occurred a sharp pain in the region of the liver, subsiding into a dull, constant ache. The patient was not aware that he had ever been jaundiced.

On admission to the Johns Hopkins Hospital on February 5th, 1901, he was found to have a much enlarged liver which presented several nodular protuberances along its lower border. The lower part of the right intercostal region showed some bulging. The percussion note was impaired and the breath sounds suppressed over various parts of the lungs. There was no œdema of feet and legs.

The diagnosis of carcinoma of the liver was made.

On March 3rd he was discharged unimproved. On April 4th he was seen at his home and found to have a marked ascites. Paracentesis being performed, four or five litres of fluid resembling pure blood were withdrawn from the peritoneal cavity. He rested well the next night, but died the following day.

An autopsy was performed 26 hours after death.

The body was rather emaciated and the abdomen somewhat distended. In the peritoneal cavity about a litre and a-half of blood-stained fluid was found, and there were blood clots in the pelvis.

[1] Ziegler's Beiträge, XVI, S. 341.

[2] Ziegler's Beiträge.

[3] Pathologische Anatomie, 1887, S. 955.

[4] Virchow's Archiv, Bd. 159, 1900.

FIG. 1.—Section of liver, natural size.

a. Large tumor nodule, broken down at *c*.
b. Fibrous tissue capsule. *d*. Fresh well-preserved nodule.
e. Necrotic tumor mass. *f*. Hæmorrhagic liver parenchyma surrounded by connective tissue bands.
g, *i*. Small tumor nodules. *h*. Areas of necrotic liver tissue in connective tissue frame-work.
j, *j'*. Tumor masses in veins. *v. c*. Inferior vena cava.

On opening the thoracic cavity, small, opaque, yellowish nodules were seen projecting beneath the pleuræ. The largest of these was 10 mm. in diameter. On section they were seen to be rounded masses of a pasty material. The main artery of the left lung contained masses and strands of the same material, and this extended into the arteries all through the lung, and was in some cases traced to the superficial nodules. The lung substance was very œdematous. No further metastases were found in the body.

The liver was enlarged and weighed 3580 grams. Old adhesions bound the organ to neighboring structures.

The capsule was thickened and the parenchyma soft in consistency. The surface was mottled dark green and yellow, contrasting with the greyish pink ground substance. There were numerous elevated yellow nodules, some 4 cm. or more in diameter, under the capsule, and several large globular masses, quite soft and fluctuant, projected from the upper surface. A similar large mass extended into the peritoneal cavity to the right of the gall-bladder. The superficial portion of this was broken down into a pasty necrotic mass, to which a blood clot was adherent. This was evidently the source of the peritoneal hæmorrhage. The inferior vena cava, on being opened, showed projecting into its lumen from the mouths of the hepatic veins masses of yellowish friable material. The largest of these reached quite to the heart. Section of the portal vein at its entrance into the liver showed it to be similarly filled with a tumor mass.

On section the liver presented a remarkably variegated appearance. The greater part of the right lobe was occupied by a large spherical tumor measuring 16 cm. in diameter. (Cf. Fig. 1 a.) Bounding this on the right was a capsule of tough translucent fibrous tissue. (b.) Trabeculæ of the same tissue ran through the substance of the mass, forming a coarse network in whose meshes lay a necrotic, sometimes ragged, material stained green with bile, or hæmorrhagic, or of a pinkish yellow color. To the left of this was a mass of firm, well-preserved, yellowish tumor tissue, also subdivided into nodules. The isolating fibrous tissue bands were here very fine. (Cf. Fig. 1 d.) This mass was seen at one point to be continuous with a portion of the tumor filling a large vein. Round about this large globular mass several smaller nodules were found, some measuring 3 or 4 cm. across. These were at times well-preserved, and again broken down and necrotic. (Cf. Fig. 1 e.) The peripheral portion of the right lobe surrounding these tumor masses was much firmer, consisting of a network of connective tissue trabeculæ in whose meshes lay a deep red rather soft material. Small greyish dots were frequently seen in this dark red substance. (Cf. Fig. 1 g.) The strands of fibrous tissue sometimes spread out into large areas, measuring at times 1 cm. across, in which lay opaque yellow dots. The presence of these connective tissue trabeculæ gave rise to the nodular appearance on the surface of the liver.

In the left lobe there were no large tumor masses. On section the cut surface showed the same nodular appearance as the outer surface, and seemed to be made up of numerous independent rounded tissue masses, held together by a framework of connective tissue. These rounded areas were partly olive green (cf. Fig. 1 h), partly of a yellowish fawn color (i), and partly hæmorrhagic and more opaque. The green and yellow nodules gave a peculiar marbled mottling to the outer and cut surface of the liver. Some areas were necrotic, soft and pasty, resembling those in the right lobe, except that they were smaller and fresher. Veins occurring in the cut surface were almost constantly filled with the soft, granular, yellow mass of the tumor. (Fig. 1, j, j'.)

The following anatomical diagnosis was made. Primary adeno-carcinoma of the liver with invasion of the portal and hepatic veins. Metastatic growths in the pulmonary arteries and lungs. Cirrhosis of the liver. Ascites. Hæmoperitoneum.

MICROSCOPICAL EXAMINATION

Liver Parenchyma.—There seems to be comparatively little healthy hepatic tissue left in the entire organ. In the rim of the cirrhotic liver lying between the large tumor and the capsule, the lobules are very hæmorrhagic. (Cf. Fig. 1, h, f.) In places where the process is not far advanced the central vein and capillaries are seen to be deeply congested and the liver cords well preserved only at the margin of the lobule. In many instances the process has seemingly gone much further, and the lobule appears as merely a mass of extravasated blood, all trace of liver cells being effaced. In some areas the cells show a compensatory hypertrophy. While the cords do not seem to deviate markedly from their normal arrangement, the cells are enlarged and irregular in form, and have frequently two or more nuclei which are likewise enlarged and take the hæmatoxylin stain very deeply. Such hypertrophied forms are described later in connection with the young nodules. (See Histogenesis.)

The nuclei of the capillary endothelium sometimes show a curious hypertrophy. They have become rounded or oval, stain very deeply with hæmatoxylin, and are sometimes so numerous as to fill the space between adjacent liver cords.

The olive green nodules which stud the left lobe prove to be necrotic liver tissue deeply bile-stained. Other necrotic areas are uncolored by bile.

The nodular appearance characterizing the whole of the left lobe (cf. Fig. 1) is due to an extensive new growth of fibrous tissue, which marks off areas of degenerated liver, as well as tumor nodules of various sizes, but does not tend to invade the individual lobules.

The fibrous bands reach their largest dimensions in the right lobe. (Cf. Fig. 1 b.) In this interlobular connective tissue one can determine an increase in "newly-formed bile ducts" which are occasionally widely dilated. The great length and narrowness which these duct-like canals sometimes show, together with the fact that they branch in a complicated way and even anastomose, seems strong evidence in favor of the view that they are really new-formed structures and not merely rows of compressed liver cells. In this instance, though they are very evidently continuous with well-defined bile ducts, their lining cells often become so flattened as to resemble thick endothelial cells. (Cf. Fig. 2.)

Tumor.—The neoplasm exhibits some diversity in structure.

The large mass (Fig. 1 a) proved entirely necrotic, so that its histological characters were obscured. Sections were made to pass through the areas marked d, h, j, g in Fig. 1, and in these the nature of the tumor could be studied. Very commonly when it is found as a nodule, and especially when it lies in a blood vessel, it consists of a mass of cords or strands lying side by side, and, as seen in longitudinal section, forming frequent anastomoses. (Cf. Fig. 3.) These cords vary in size, some consisting of but 5 or 6 cells in cross

section, others containing 50 cells or more. They are clothed by a delicate flat-celled endothelium. (Fig. 3 b.) A lumen, central or excentric, is occasionally seen—a mere gap between the surrounding cells, having no endothelial lining. (Cf. 4 c.) No bile was noted in these tubes, as was found by v. Heukelom.[5]

The spaces between the cord-like structures very frequently contain red blood corpuscles. (Cf. 3 c.) This phenomenon is easily understood in the case of the intravascular growths, where such spaces represent the lumen of the vessel. Where a tumor nodule is found in the midst of hepatic tissue, we may conceive that the blood spaces constituting the capillaries of the liver have persisted between the strands of tumor cells which spring from and correspond with the liver cords, as is described below under histogenesis. This conception explains also the origin of the endothelial covering of the strands. We have here the capillary endothelium which normally lies next to the liver strand.

Nodules of this description, varying from 2 or 3 mm. in diameter to a microscopic size, stud the left lobe, where they sometimes lie within a vessel wall, and again are immediately surrounded by liver cords. Transitions from liver cords to tumor columns were found in this region. (See Histogenesis.) The nodules here are in the main well-preserved. The small whitish dots (Fig. 1 g) consist of this cord-like form.

The well-preserved nodule (Fig. 1 d) gives a picture of larger and smaller masses almost invariably of this strand-like form, separated from one another by fine bands of connective tissue. There is occasionally a central necrosis, but in general the tissue is well-preserved here.

A second form exhibited by the tumor is somewhat more complicated. In this we seem at first sight to have a tissue made up of a very indefinitely arranged mass of cells. Closer examination reveals, however, elongated and irregular spaces (cf. Fig. 5 a) lined by a delicate endothelium (b) and containing blood corpuscles and fibrin (Weigert's fibrin stain). These spaces are further bounded by a row of cells, either polygonal and irregularly placed, or cylindrical and arranged side by side. Adjoining this row is a mass of irregularly arranged cells, beyond which one sees another row of polygonal or cylindrical cells, which, with a layer of endothelium, bounds another blood space.

Some of these spaces are not entirely walled off, but are continuous with the spaces described above as lying between the tumor cords. One may then conceive this part of the tumor to be made up of strands of cells branching and anastomosing very freely, and thus sometimes enclosing blood spaces with intact endothelium. The marginal cells of such cords generally retain a definite arrangement, while the central ones are rather disordered. Areas adjoining such a field as has been just described frequently show an absolute irregularity in the appearance and disposition of the cells. Giant forms with a single immense nucleus or with many large nuclei occur in such areas (cf. Fig. 7), and an extensive fatty degeneration may be exhibited by all the elements, giving a spongy appearance to the entire field.

In the intravascular growth both forms of the tumor are seen, as well as the irregular areas with bizarre giant cells. Caseous tracts are also found here, showing sometimes faint outlines of tumor cords or lines of intact endothelium.

The vessel wall has its proper endothelium preserved, and that part of the tumor lying in apposition is similarly clothed. (Cf. also Fig. 6.)

Fragments of greenish bile are found in abundance both in the tumor cells and lying free in the spaces between them.

Tumor Cells.—In the strand-like form of the tumor (cf. Figs. 3, 4, 6) the cell body, as compared with that of a normal liver cell, is small, the nuclei in a tumor cord appearing more closely crowded than those in a liver strand. The shape of the cell is also modified, being moulded by the pressure of its neighbors to a polygonal outline. The protoplasm has the granular appearance of a liver cell, and in general is not bile-stained. The nucleus resembles that of a liver cell in size and appearance; is round or oval and contains many minute points of chromatin irregularly scattered and often joined together by fine strands. A larger mass of chromatin is frequently seen towards the centre. (Cf. Fig. 4.) Sometimes the cells in the outermost row in a cord, those lying next under the endothelium, have a denser cytoplasm and a more solid-looking, deeply-staining nucleus. (Cf. Fig. 6 d.)

With Mallory's stain the reddish-blue protoplasm has a vacuolated, granular appearance. The nucleus is provided with one or occasionally more oval or round bright red bodies containing vacuoles of relatively large size. At times the whole nucleus seems to be stained red, owing to the large size of this nucleolar body. In other instances, however, there is a space between the nucleolus and the nuclear membrane in which appear dots and strands taking the reddish-blue stain of the cytoplasm and the nuclear membrane. A hæmatoxylin stain does not differentiate between the various nucleolar bodies; all are stained blue.

In the second variety of the tumor (Fig. 5) much more irregularity in cell structure is seen. The cells bounding the blood spaces are frequently cylindrical in form and have oval or elongated nuclei (Cf. Fig. 6). The giant forms found here have already been referred to (Cf. Fig. 7). The nuclei in these cells are generally very dense, showing large irregular deeply-staining blocks against a ground filled with fine chromatin particles.

In portions of the tumor undergoing degeneration, the cytoplasm is often replaced by a fat globule, while the nucleus is very dense and assumes bizarre forms.

In certain of the young, fresh nodules seen especially in the left lobe, the nuclei show mitotic figures.

Metastases.—In the larger and smaller branches of the pulmonary arteries masses of the tumor are found as noted on naked eye examination. In the smaller vessels the growth is in general of the cord-like form and is fairly well preserved (Cf. Fig. 6). In the larger arteries the second

[5] Loc. cit.

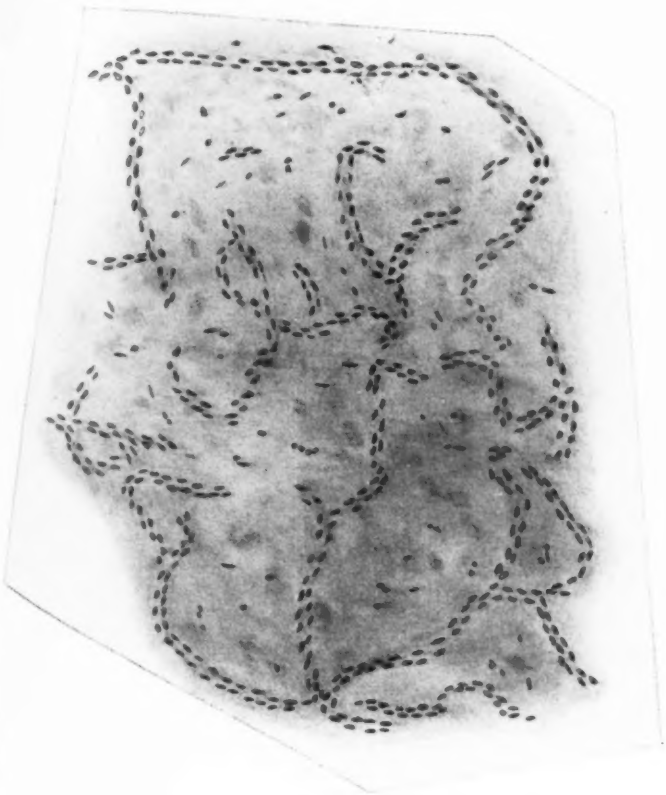


FIG. 2.—Connective tissue showing newly formed bile ducts. ×112.

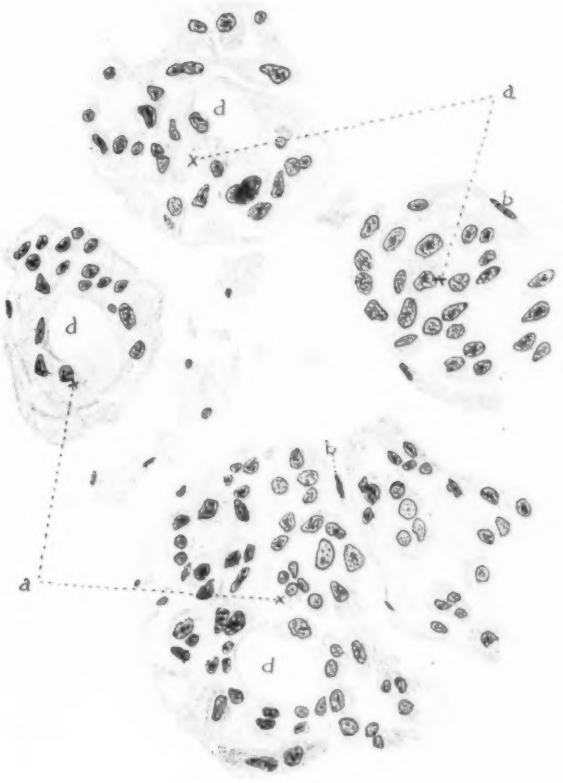
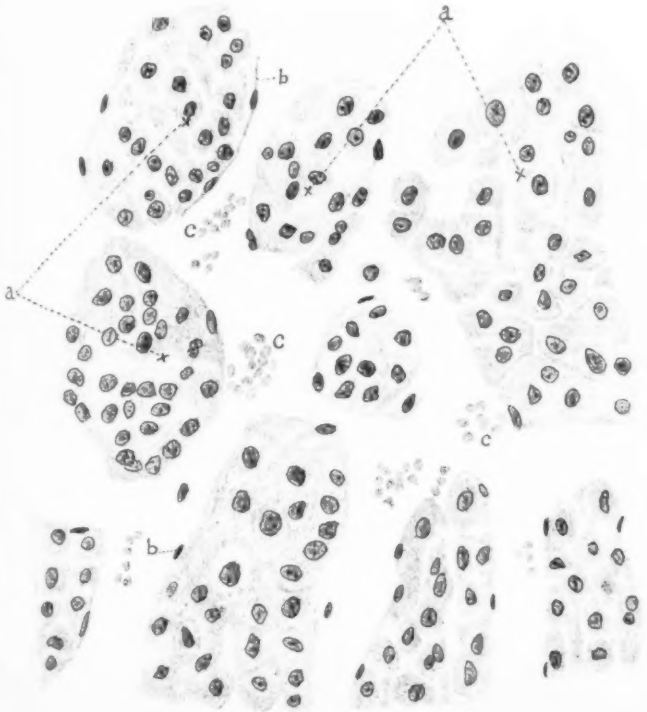


FIG. 4.



FIGS. 3 and 4.—Tumor. First form. ×450.
a. Tumor cords. *b*. Endothelium. *c*. Red blood cells.
d. Lumen.

FIG. 5.—Tumor. Second form. ×85.
a. Blood spaces containing fibrin and red blood cells.
b. Endothelium. *c*. Cylindrical cells.

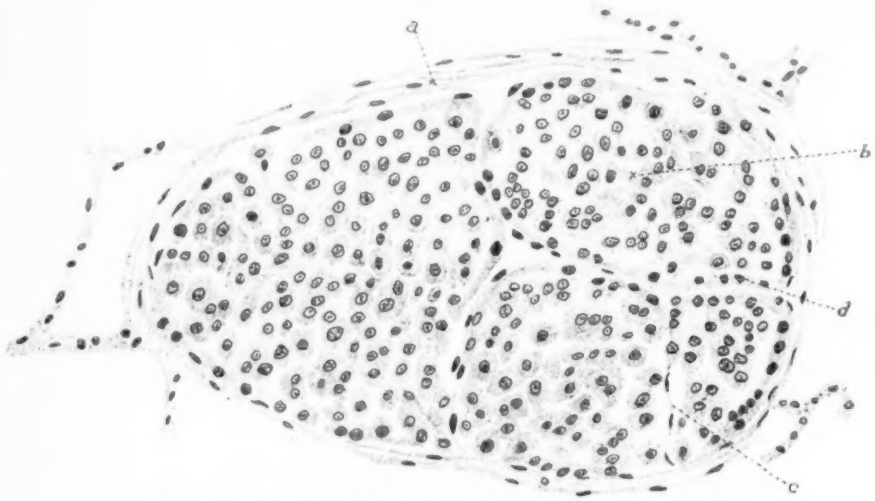


FIG. 6.—Tumor nodule filling pulmonary vessel × 112.
a. Vessel wall. *b.* Tumor cord. *c.* Endothelium.
d. Dense nucleus in marginal cell.

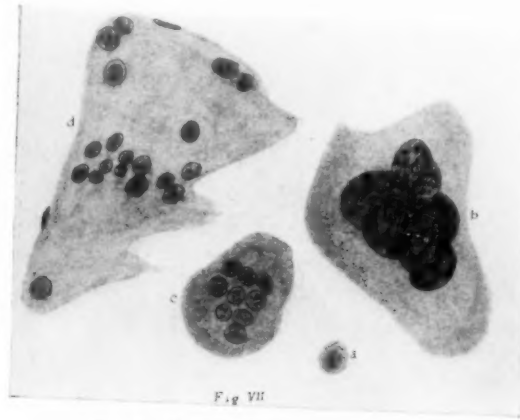
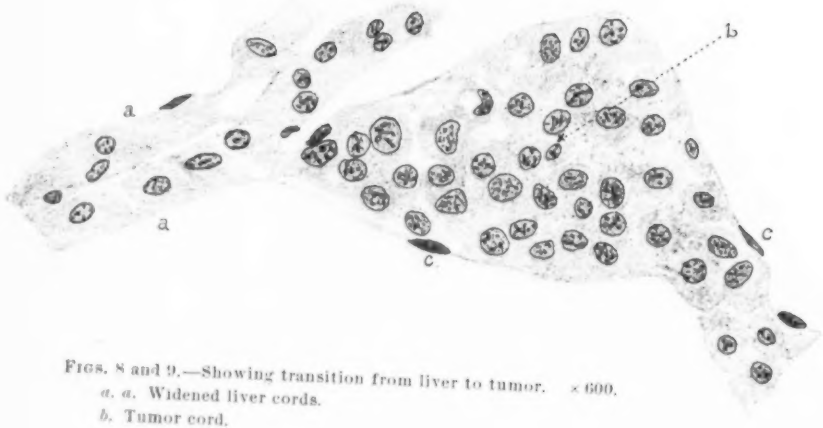


FIG. 7.—Cell forms from tumor. × 300.
a. Ordinary tumor cell.
b. c. d. Multinucleated giant forms.



FIGS. 8 and 9.—Showing transition from liver to tumor. × 600.
a. a. Widened liver cords.
b. Tumor cord.
c. Endothelium.

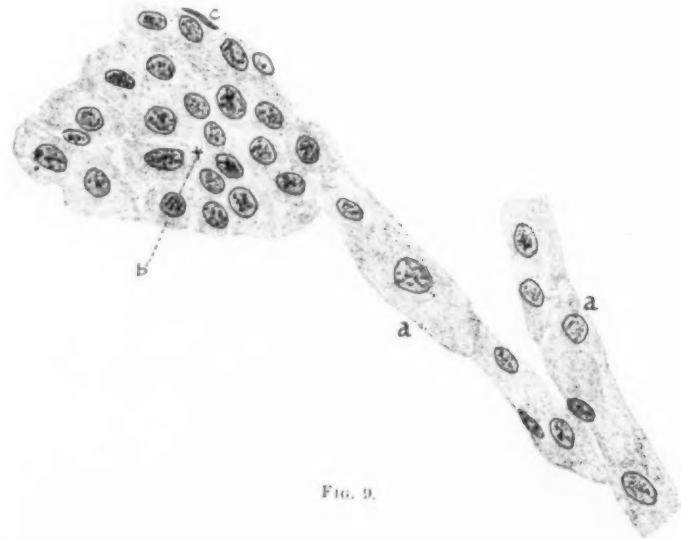


FIG. 9.

form (Fig. 5) is also found; and here again disintegration and irregularity in the arrangement of the cells, and fatty degeneration are observed. The vasa vasorum in the walls of the larger vessels are frequently filled with the strand-like growth, and occasionally the tumor is found growing in the alveoli adjoining the vessels, sometimes forming large nodules.

Cloin[6] in his case found bile in the metastatic growths in the lung; none was observed in this instance.

Histogenesis.—This is best traced in young nodules (Fig. 1 h). The ground substance in which the nodules lie, the liver tissue, shows in the main such marked caseation that it is difficult to get an idea of its structure. The cells surrounding the nodules are generally in good condition, however. While it is not made out that the liver lobules become definitely enlarged, the cell-cords in the neighborhood of an area of new growth exhibit distinct hypertrophy, and in many instances the relation between liver cords and tumor strands can be traced. The nodule seems very often to have grown so exuberantly as to compress the cords of cells about it, giving them a squeezed appearance and concentric arrangement. The transition from these cords to the structures of the new growth is as follows:

The cells in the surrounding cords become larger, their nuclei are also enlarged and take a deep hæmatoxylin stain, and this widened atypical strand becomes still wider, comes to have several cells abreast, and passes thus gradually over into one of the cords definitely belonging to the tumor (Cf. Figs. 8 and 9).

v. Heukelom[7] gives exquisite pictures of just such a transition as that seen here, and Schmeiden[8] and Cloin[9] note a similar phenomenon.

Proliferation of the tumor cord thus produced can be readily conceived to give rise to nodules of larger and smaller dimensions as they are found all through the left lobe. Through anastomosis of adjacent cords the more complicated variety of the new growth may arise, as suggested above. The blood capillaries of the liver are evidently continuous with the spaces between the different tumor strands. The manifold small nodules scattered through the liver are then not of metastatic origin, but are primary growths derived by a direct transformation from the liver cells.

A word may be said here on the connection between cirrhosis and adenoma formation.

A few authors hold that the two processes are quite independent of each other. Many others think the coexistence of the two lesions is too frequent and too typical to be merely fortuitous. Some consider the adenomatous growth the primary disturbance, the cirrhotic process being dependent thereon. On the other hand many hold that the cirrhosis is antecedent and the tumor growth a result. Owing to the destruction of liver parenchyma, due to an extensive new growth of connective-tissue, those liver cells which are intact undergo a vicarious hypertrophy. The large atypical cells thus produced are transformed in some way into adenoma cells.

A very rational view is suggested by the work of Marchand. That author found that after extensive destruction of the liver cells by some unknown poison (acute yellow atrophy) a connective tissue new growth accompanies the efforts at regeneration of the liver. These efforts are of two or perhaps three kinds, according to him: 1. The formation of large, irregular lobules from the lobules or portions thereof remaining. 2. The proliferation of cells—even degenerated ones—in the greatly degenerated areas. 3. The growth of bile ducts into the lobule.

In these cases of carcinoma of the liver the course may have been analogous. There has been a destruction of the liver tissue by some poison, to so great an extent that the organism is forced to undertake efforts at regeneration. These consist in the formation of atypical cell masses; and a further more atypical development of such masses results in the production of a malignant growth, the exact changes at this border-line between compensatory hyperplasia and malignancy being obscure to us. The cirrhosis is thus a phenomenon of invariable occurrence as part of the healing process after the degeneration of the liver, but is of quite secondary interest in the production of the tumor.

Orth[10] writes "One cannot help asking whether we have not here a vicarious hypertrophy which from some unknown cause proceeds past its goal and by atypical growth leads to the formation of a tumor," and Schmieden[11] in a general way holds the same opinions.

As conclusions from the foregoing study we may state:

The case shows a multiple, primary neoplasm in the liver, accompanied by a widespread interlobular cirrhosis.

The new growth has invaded both portal and hepatic veins, and is found filling the pulmonary arteries and forming metastatic nodules in the lung tissue.

The tumor shows some variation in its structure, and transitions from liver cords to tumor strands are demonstrable.

The malignant tumor possibly results from the progressive atypical growth of hyperplastic masses produced coincidentally with the cirrhosis by a reparatory and regenerative process, after the extensive destruction of the liver by some unknown poisonous agent.

[6] Prager Medicinische Wochenschrift, XXVI. Jahrgang, S. 261, 275.
[7] Loc. cit. [8] Loc. cit. [9] Loc. cit.
[10] Loc. cit. [11] Loc. cit.

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CONCERNING AN IMPROVED METHOD OF MAKING COLLODIUM SACS.

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(*From the Pathological Laboratory of the Johns Hopkins University and Hospital.*)

It will not be too presumptuous upon my part to state that by the majority of workers in our bacteriological laboratories both the utilization and the making of the collodium, or celloidin sac for experimental purposes is almost wholly neglected. This is largely to be accounted for, perhaps, either by the lack of a fully described technique of a reasonably easy method, or by the presentation of a too cumbersome method which entails spending too much time in overcoming many difficulties of a technical nature, thus naturally leading to discouragement and the abandonment of further attempts. It is, therefore, the object of this paper to present an improved method of making these sacs, the technique of which is simple and readily acquired, whilst the materials are practically always at hand in every laboratory for making them upon briefest notice.

Before entering upon details it may not, perhaps, be out of place to give a short review of the history connected with the introduction of collodium sacs and the several methods employed in their manufacture, so far as I have been able to get at the facts.

Taking advantage of the facilities for osmosis afforded by collodium films, the idea of sealing up bacteria in the living state within such films and placing them within the animal body to obtain, if possible, the effects of the elaborated diffusible toxins without the presence of the germs themselves in the blood or tissues, presented itself some six or seven years ago to Drs. Metschnikoff and Roux of the Pasteur Institute in Paris. This idea was successfully wrought out by them in their studies upon the toxins of Asiatic cholera in 1896, and later by Nocard and Roux who by this means undoubtedly isolated the causative factor of pleuropneumonia of cattle. This latter study was a veritable triumph, inasmuch as it *proved* what had heretofore been only suspected, that in certain infectious diseases we had to deal with living matter of such minute proportions as to be beyond the ability of the most powerful microscope to define.

Furthermore, Nocard was able by means of the sacs to transform the characteristics of the human tubercle bacillus into those of the avian variety, thus adding proof to the theory of the mutability of closely allied forms of bacteria.

It was also demonstrated that the virulence of pathogenic bacteria could in some instances be highly exalted by implanting a successive series of collodium sac cultures in the peritoneal cavities of animals.

Quite recently, McCrae has pointed out the value of the sac in the classification of bacteria of closely allied species, and produced agglutinative reactions in the blood of one animal inoculated with several sacs, each containing a different species of bacterium.

In perusing the writings of Roux, Metschnikoff and Nocard relative to their experimentations with collodium sacs, one curiously enough fails to glean the least hint as to how these sacs were prepared, nor do later publications from the Pasteur Institute seem to contain any references to the technique.

In this country some years ago (1898?) partially successful attempts were made by Dr. E. L. Trudeau of Saranac Lake to turn out a serviceable celloidin sac. He was, I believe, the first to suggest the employment of the ordinary gelatin capsule as a mold upon which to form the sac. The method used was found not to be sufficiently well developed to yield satisfactory results and publication was never made, although Dr. Trudeau openly spoke of his technique to many.

Dr. T. M. Prudden of Columbia University about the same time developed a method of making celloidin sacs by using a tube in whose bottom a small hole was blown, which previous to dipping was sealed by a thin film of celloidin. The tube was dipped in the medium and rolled until it had set, when the sac was dislodged by blowing into the tube, the air escaping from the small hole in the bottom and dissecting the film away from the sides of the tube. The method did not prove very satisfactory, nor did the coating of gelatin capsules in several ways yield any better results.

To Professor F. G. Novy of the University of Michigan belongs the credit of being the first to publish the method of Metschnikoff and Roux. This consists, briefly, in coating a glass rod of a certain thickness with collodium, several times, until a sufficiently thick layer is deposited, allowing it to set and then peeling the sac off by inversion, trimming it and affixing a piece of glass-tubing of proper calibre by the aid of gentle heat. By further manipulation the capsule is filled with broth, sterilized, inoculated and the tube sealed up close to the capsule by means of the gas flame.

Utilizing Trudeau's idea of employing the gelatin capsule as a mold, Dr. John McCrae of Montreal has devised a most successful method. His technique is briefly stated as follows: the larger end of a selected gelatin capsule is made to adhere to the end of a piece of glass-tubing of similar bore, by heating the latter a little. The capsule is then dipped in thin celloidin several times until a sufficient coating has formed, when it is further hardened by immersing in cold water. The gelatin is next dissolved away by hot water, the resulting celloidin sac filled with broth, sterilized, and preserved in a broth culture tube for use when desired.

Quite recently Dr. C. S. Gorsline of the University of Michigan has published a method which is essentially that of Prudden, but he has overcome difficulties of dislodging the sac from the glass tube by filling the latter with water

and forcing the water out through the hole by blowing into the tube. The sac is then fitted to a suitable piece of glass tubing by gently heating the latter, and the final technique is that of filling with broth and sterilizing under conditions suitable for preserving the capsule from contamination until required for implanation.

Regarding the method which I have devised, I can make few claims to any originality, having built upon Dr. Trudeau's idea of using the gelatin capsule, and benefited also by McCrae's efforts in the same line.

The procedure is carried out as follows, having first assembled the necessary materials consisting of—

(*a*) Empty gelatin capsules:—those of Parke, Davis & Co., may be had in the market, of almost any size desired, ranging from "No. 0—medical," holding about 1 cc., up to "No. 10—veterinary," holding approximately 30 cc.

FIG. 1.

FIG. 2.

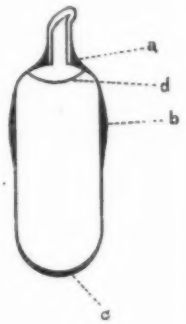

FIG. 3.

FIG. 1.—Empty gelatin capsule, actual size. "No. 12 Veterinary," P. D. & Co.

FIG. 2.—Glass tube sealed into end of capsule.

FIG. 3.—Sac ready for insertion. *a*, *b*, *c*, points of reinforcement; *d*, limit for height of the column of fluid inside.

(*b*) Glass-tubing:—preferably that having a gross diameter of from 2.5 mm. to 4 mm., or if such sizes are not at hand, coarser tubing may be drawn out to suit the purpose.

(*c*) Collodium (U. S. P.), or Celloidin:—either solution to be tolerably thick and kept in a small round jar, with closely fitting top, to the depth of 4 to 5 cm.

(*d*) A Drying-rack made by piercing a half inch pine board with rows of long fine wire nails at intervals of 4 cm.

(*e*) A glass-blower's burner, or some device to secure a long narrow flame.

(*f*) A small file.

Procedure.—Having selected the gelatin capsule, a piece of glass tubing 4 cm. long and of 3 mm. diameter is taken and the sharp edge of one end is filed down and that end heated a moment in the flame of a Bunsen burner, then the unseparated capsule is taken in the grasp of the left thumb and fore-finger, and with those of the right hand gentle pressure is made with the heated end of the tube directly against the centre of the end of the smaller portion of the capsule until the tube projects within the cavity not more than 2 mm. (see Fig. 2). In a moment it becomes fast, if the heat has not been so great as to burn the gelatin, then one proceeds to apply a coating of collodium by means of a match or platinum loop around the junction point of tube and capsule. Now separate the capsule, and by means of a knife or other sharp-pointed tool clear the bore of the tube of the hardened cap of gelatin, then replace the two sections as before.

We now have the gelatin capsule centrally fixed to a permanent spindle which renders the coating with collodium or celloidin an easy matter. Before proceeding, however, to the coating process see that the collodium previously applied to the neck is quite dry. This being so, the jar containing the collodium is held tilted at an angle of about 50°, the gelatin capsule is slowly lowered into it and submerged sufficiently to coat the tube to the height of 1 cm. above the end of the capsule. It is now brought slowly up out of the collodium in the horizontal position quietly rotating it at the same time, care being taken not to remove too much collodium else the smoothness and evenness of the film will be interfered with.

Now rotate continuously until it becomes set, slowly at first to ensure an even distribution of the material, then more rapidly, blowing upon it occasionally to hasten the setting. I do not believe that immersing the capsule in water at this stage tends to any advantage, rather the reverse, for opacities are liable to develop in the film which may become permanent and spoil the transparency of the finished sac, and may be, prevent proper dialysation. When the film has set, the capsule may be placed at once upon one of the nails of the drying-rack to dry. It is of especial importance to let it dry out thoroughly. After this process is ended it is not necessary to repeat the coating unless the collodium is quite thin (I might say, here, that before using, I allow it to evaporate down to the consistency of a moderately thick syrup), for the thicker the film the less useful a sac becomes, however, after a trial or two each worker can use his own judgment as to what is enough. It is necessary at this stage to reinforce the sac at certain points, but before doing this it may be well to wind a piece of silk thread on the glass tube close to the capsule, tie it and leave two ends hanging so that the sac later on may be readily handled. Referring to Fig. 3, *a*, *b*, *c*, the points to be reinforced are indicated and the measure is carried out by means of the platinum loop, except in reinforcing the end which is accomplished by simply dipping the capsule again into the collodium. The sac is again placed upon the rack to dry, which occupies a short time, and it is then ready for the next step. In this way ten or twelve capsules may be set up in succession, and usually by the time the last one is coated the first will be found ready to be reinforced, and upon strengthening the last sac the first will be ready for finishing.

The gelatin must now be removed from the interior of the formed sac. This may go hand in hand with the sterilization, or constitute a separate procedure. Considering the

former proposal first, the coated capsule is removed from the rack and filled with broth by means of a Pasteur pipette, conveniently bent at right angles a little distance above the bulb, and at once placed in a broth culture tube, glass spindle downwards, and covered with broth to the extent of 1 to 2 cm. above the capsular end. The tube now holding the sac is autoclaved for five minutes at one atmosphere pressure and 120° C., thus rendering it completely sterile. It will be found upon removal from the autoclave that the gelatin has been melted and expelled from the sac and has gone into solution with the broth, so that we have now the sac filled with a medium of weak nutrient gelatin, which may congeal on cooling, constituting an excellent pabulum for bacteria.

If, however, it is thought to be desirable to remove the gelatin from the sac, the latter is to be filled as before, placed in a dish of hot water, when after a while the gelatin melts and may be washed out by means of the pipette, the process being repeated as often as is necessary. The sacs are then to be sterilized in the manner stated in the foregoing, and may be preserved in good condition for use at any time by leaving them in the culture tubes.

By the foregoing processes it will be found that very little, or no shrinking occurs in the sac if pains have been taken to dry it in the air after coating, and if the manipulations in coating have been deliberate no bubbles should be seen in the walls of the sac. Likewise, the glass spindle remains firmly fixed in position requiring considerable rough handling to cause its separation. It is surprising how tough such a sac can be after only one application of the thick collodion, and at the same time appear so frail.

As Novy points out, some varieties of collodium and celloidin are of little value for the manufacture of sacs on account of having such a low index of dialytic power, or are even without any, so it would be well to make a test of the osmotic properties of a sac before making a number of them.[1] This test can best be made by completely removing the gelatin and filling the sac with a saturated solution of magnesium sulphate, immersing it in distilled water and testing the latter for the presence of the salt at intervals.

The technique of inoculating and closing the sac will now be discussed. The culture tube containing the sterilized capsule is to be emptied of its fluid contents and the sac to be then deposited in a sterile Petri dish or some other suitable vessel. With a pair of sterile dissecting-forceps the glass spindle will be taken hold of and the sac held vertically upwards, whilst with a Pasteur pipette a small quantity of the contents is removed (see Fig. 3, *d*) so as to obviate any chance of the fluid within flowing up into the tube during the sealing of the glass, then with the pipette a small portion of the inoculating material is very carefully deposited within the capsule, caution being needed not to permit any to touch the sides of the tube.

The sealing of the glass tube demands most delicate manipulation upon the part of the operator. Having prepared the glass-blower's burner, the spindle of the sac is taken hold of high up with another pair of sterilized forceps, and with the first pair (heated a little to disperse moisture from inside the tube near the site of sealing) a fresh grasp is to be taken lower down close to the junction of the sac and the tube, then with the sac held vertically the glass tube is brought into contact with the narrow non-luminous flame of the burner a short distance above the lowermost forceps, precautions being taken to first drive off any moisture which might be lodging at or near the point of sealing, the glass rapidly fuses and is drawn apart gently and the end attached to the sac sealed perfectly, as shown in Fig. 3. Care must still be exercised until the glass point is well cooled lest any agitation of the sac cause its contents to be thrown up into contact with the glass and crack it and render the sac useless. This danger being past, it is well to wash the sac off with sterile water or broth and replace it in a sterile broth culture tube until abdominal section has been performed upon the animal when it is removed and cautiously inserted and gently pushed away from the site of the incision by the sterile finger of the operator. McCrae very wisely suggests that at first the operator should incubate the inoculated sacs for 24 hours in broth to assure himself that there is no flaw in his technique of making the sac, and if the broth remains sterile then to proceed to insert the capsule in the animal; this procedure may be abandoned if it is seen that one has acquired the technique perfectly.

Resume of Technique.

(*a*) Preparation of the glass-tubing.
(*b*) Fixation of same to gelatin capsule by heat.
(*c*) Coating of junction-point while warm.
(*d*) Clearing the bore of tube of gelatin.
(*e*) Coating of the whole capsule.
(*f*) Air-drying on the rack.
(*g*) Reinforcing, and again drying.
(*h*) Charging the capsule with broth.
(*i*) Sterilization in autoclave (removal of gelatin).
(*j*) Inoculation.
(*k*) Sealing up glass tube.

Advantages of the Method.

1. There are practically no limitations to the size of the gelatin capsules requisite for experimentation.

2. There is no necessity to remove the sac from the mold, which is dissolved and forms a nutrient medium for the growth of the inoculated organisms.

3. A greater relative amount of dialysing surface is obtained by the use of a minimal quantity of glass, than is gotten by other methods.

4. By completely air-drying the collodium, a thinner, tougher, less shrunken and an equally good dialysing mem-

[1] I am, however, inclined to believe that the condition is more often caused by the water-hardening process, or by having too thick a film, than by the quality of the material.

brane is obtained than by the water-hardened, rod or tube systems.

5. The method seems to throw fewer discouraging difficulties in the way of the operator than most of the other schemes.

That the method is not perfect will occur to all, but I offer it in the hope that it will afford some the opportunity of accomplishing with less difficulty what has heretofore been one of the most involved of bacteriological technical procedures, and also the hope that it may finally lead some one to suggest a simpler and a more perfect method.

I take this opportunity to express my thanks to Doctors Trudeau, Prudden and Gorsline for information regarding their own technique.

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PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS HOSPITAL MEDICAL SOCIETY.

Meeting of November 18, 1901.

Dr. Osler in the chair.

Arthritis Deformans. DR. BOGGS.

This man, 45 years of age, came in November 6, complaining of stiffness of the hips, back and neck. His family history is quite interesting in that his grandfather suffered with rheumatism and was disabled with stiff joints as was also his father and one paternal uncle. One sister also had rheumatism and tuberculosis, one of the etiological factors mentioned by Marie in this disease. His past history shows that measles was his only acute illness until he was grown up but he had lues at the age of 23; has never been addicted to the excessive use of alcohol.

His attacks began at 12 years of age with what he calls muscular rheumatism, that is, the joints were not affected but he had pains in the arms and legs near the joints without limitation of movements. By the time he was 15 he was unable to play in outdoor sports with the other boys because cold air brought on these attacks. These would sometimes be very severe for two or three days but he was not compelled to go to bed with them. When 24 years old he had his first severe joint attack, beginning in the right knee and extending down to the foot. Then it began in the left knee and foot, then changed back to the right and finally back to the left again. During this attack he lost 30 pounds in weight but in the course of three months was able to go about without the use of a stick. Shortly after this he noticed a stiffness and spasm in the abdominal muscles which gradually drew him over into a flexed position and his waist became so small that he could span it with his two hands. This condition lasted for 15 years and about the time of the involvement of the abdominal muscles he began to have pain and spasm of the muscles of the eyes. These have persisted and at times are quite severe. During an epidemic of influenza in 1891 he had an attack, probably of acute articular rheumatism in which the feet and the hands became very much swollen. He has been getting worse since, the swelling being followed by pain and then after the acute stage by an increasing stiffness of the neck. At times there has been difficulty in swallowing. In 1894 he had another attack, this time affecting the hips, thighs and small of the back, the stiffness in the back increasing gradually until by 1896 the neck and back were rigid but there was slight motion still at the hips. There have been no acute attacks since but by 1898 he had lost entirely the power to flex the hips and assumed the characteristic position he now exhibits. Between 1896 and 1898 he noticed an increasing stiffness of the lower maxilla and at times he could not open the mouth sufficiently to take food, he used vigorous massage, however, prying his mouth open by one means or another and has regained some degree of motion of the jaws. He has no motion whatever in the spine now and the only motion left in the hip joint is a very slight degree of abduction when the legs are firmly pressed apart. The knee joints and ankles are perfectly free and he gets about with a fair degree of comfort but has difficulty in going up or down stairs. He is able to carry on his business as a lawyer.

The case is an illustration of one of the types of arthritis deformans described by Strümpell and Marie in which the spine is affected and sometimes the hips and shoulders. It is notable that the patient has none of the nodes of Heberden so common in another group of cases.

Discussion.

Dr. Osler.—This is one of an interesting group of some 5 or 6 cases we have had illustrating spondylitis deformans. This case in particular shows that the affection is only the spinal variety of arthritis deformans. This man has had recurring attacks of arthritis for many years. He has not the usual extreme nerve root pains but I judge the attack he had some years ago was due to pressure on the nerves as they passed through the foramina. Some of the cases have agonizing pains. One point that Dr. Boggs did not mention is the condition of complete anchylosis of the thorax which is a constant feature of this form.

Anatomically this is the oldest known disease. Skeletons with spondylitis deformans have been found among the mummies of Egypt.

Dr. Tinker.—The surgical side of rheumatoid arthritis and arthritis deformans seems to be worthy of consideration because it offers the only possible means of relief for a very distressing deformity. Very little has been written about it, but from some recent reports it would appear that the condition is not quite as hopeless as it was formerly supposed to be. König has recently reported his experience in the treatment of 20 cases of this kind, with operation in four cases in which the hip was entirely anchylosed, as is the case here. He did resection with satisfactory results, all the patients being able to go about and at least two being able to take up their former occupations. Küster has also resected such joints with fairly satisfactory results. Some other writers have not been so fortunate and one states that he resected a joint which was quite painful with relief of the pain but without the patient being able to go about. In this country very little has been done in an operative way. Goldthwait, of Boston, has operated upon two cases with fairly satisfactory results by breaking up the anchylosis and following that with massage. In the earlier stages of the affection massage is probably beneficial and I believe this man has benefited himself very much by his exercises. After there is firm anchylosis the Germans believe that resection is the only thing to do.

The Question of the Operative Treatment of Acute Pancreatitis. Dr. Bloodgood.

(To appear later.)

A Case showing the Deformities of Chronic Gout. Dr. Futcher.

The patient I have to show is one manifesting very advanced deformities in the joints as the result of chronic gout. The man is 54 years of age and was admitted the first time in 1894 complaining of pains in his feet, ankles and hands. There was no family history of gout. The patient had been a brick-layer most of his life but had been a brass-worker for a time after the onset of his gout symptoms. The first joint affected was probably one of the metatarso-phalangeal joints. This occurred about 14 years ago. Every year from that time up to the date of admission he had repeated attacks of arthritis, involving at times also the elbows or the knees. He had been a heavy beer drinker, 2 or 3 pints a day and occasionally a glass of whiskey. The fact that he had been a brass-worker was probably of no special importance but whether or not lead was an etiological factor in his case it is difficult to say. For several years his joints have been markedly deformed and he has been incapacitated for work.

On admission to the hospital in 1894 he had an acute attack involving the hands and feet and there were quite marked deformities of the joints, but the nodules about the joints at that time were not definite enough to be called tophi. The first diagnosis was acute articular rheumatism with a question as to the possibility of gout. Since then he has been admitted 11 times, 10 times on the medical and one on the surgical side. The attacks of arthritis have been repeated and severe and the deposits about the joints have gradually increased in size. In 1897 he was admitted with a large swelling in front of the left patella. It was found that the prepatellar bursa was inflamed and required opening. On his second admission the nodules about the joints had become more definite and there were tophi in the ears. Microscopic examinations of these showed the characteristic acicular crystals of biurate of sodium and definitely established the existence of gout. As you see there are now enormous nodules over the knuckles of both hands. Occasionally these tophi suppurate and discharge their chalk-like contents. An interesting point in this case is that, as you see here on examination, there is a large collection of tophi over the sacral region.

As to the etiology, in this case, beer is probably the most important factor. There are four marked etiologic factors in gout: heredity in from 50 to 60 per cent, alcohol in a large percentage of cases; over-eating with insufficient exercise; and lead poisoning. Of our 32 cases of manifest gout, in a total of 14,000 cases seen in 12 years, practically all have given a history of alcoholism, fermented liquors—beer, ales and wines—playing a more prominent part than distilled liquors. It is rather interesting that ten of our 32 cases have given a history of having worked in connection with lead, a percentage which corresponds closely with Garrod's statistics. All of our cases have been in males and all in white patients. In 16 the diagnosis was established by the existence of tophi and in the others largely on the history and the fact that the attacks began with involvement of the toe joints. It is interesting that three of the cases, when first admitted, were diagnosed as acute articular rheumatism and it was only on subsequent admissions that the true diagnosis was established by the development of tophi in the ears or some other part of the body. Two of these had nodules beneath the skin which were at first considered to be the subcutaneous fibroid nodules of rheumatism, but which later proved to be tophi.

There have been three cases of gout with bursitis, two over

the patella and one over the olecranon; two suppurated and were operated on.

As to the definite disturbance of metabolism which is at the bottom of gout too little is known and it is impossible to go into the various theories at present, but it is in some way connected with the disturbance of uric acid metabolism and many adhere to the theory offered by Sir William Roberts some years ago. According to Bence-Jones and Roberts uric acid does not circulate in the blood as such but in the form of a quadriurate which is very soluble and easily eliminated. In gout, however, there is defective elimination or an excessive formation and the latter accumulates in the blood, in a medium rich in sodium carbonate. Eventually a molecule of the quadriurates is broken up into two molecules of acid biurate. The sodium biurate is very insoluble and is deposited in those tissues of the body where the circulation is poorest, the temperature lowest and the percentage of sodium chloride highest; in other words, in the cartilages and periarticular structures. This seems to be the most satisfactory explanation of the disturbance of metabolism in gout but the disease is still but poorly understood.

A Case of Hæmorrhagic Pancreatitis. The Occurrence of a Fat-Splitting Ferment in the Urine. DR. OPIE.

The patient, a man 28 years of age, had before his fatal illness been in good health. There was no history of any previous digestive disturbance and he had never been jaundiced. His illness began four days before death with an attack of nausea and vomiting coming on shortly after a meal. About an hour later he was attacked with agonizing pain in the epigastric region, and the vomiting continued during the night but in the morning became less severe. The following day he suffered with violent hiccough, which continued until the time of operation. When admitted to the hospital he seemed very ill and vomited almost continuously. Examination of the abdomen showed nothing definite except distension in the neighborhood of the umbilicus. An exploratory laparotomy was performed by Dr. Mitchell and innumerable foci of fat necrosis were found studding the omentum. From this condition acute pancreatitis was believed to exist and an incision was made through the great omentum, between the stomach and transverse colon. The lesser peritoneal cavity was found to contain bloody fluid and the pancreas was covered by a coagulum of blood. It was not possible to obtain any evidences of a calculus in the gall bladder or in the bile passages and the abdomen was closed, a gauze drain being inserted in the upper angle of the wound. After the operation the patient vomited almost continuously—at first a dark brown fluid and later almost pure blood. He died on the fifth day of his illness, twenty-four hours after the operation.

An examination of the abdomen through the wound was permitted and it was possible to remove only the pancreas with the gall bladder and a portion of the duodenum. The pancreas is of great size, weighing 190 grms., the normal weight being only 90 grms. The head and tail of the organ are firm and compact in texture and yellowish-gray in color, but the greater part of the body shows a hæmorrhagic lesion. Here the tissue is soft, of mottled red and reddish-black color, without the normal gland lobulation and has the typical appearance of hæmorrhagic pancreatitis. No gall stones were found in the gall bladder or gall ducts and the duct of Wirsung was not dilated. The parenchyma is the seat of widespread necrosis in the neighborhood of which the interstitial tissue is infiltrated with red blood corpuscles, polynuclear leucocytes and fibrin. The capillary vessels at the margin of the necrotic area shows the condition of hyaline thrombosis, observed in a case previously reported.[1]

Acute hæmorrhagic inflammation, as has been shown by Dr. Flexner and others, can be produced experimentally by injecting a variety of irritant substances, gastric juice, various acids and alkalies, suspension of bacteria, etc., into the pancreatic duct or into the parenchyma of the gland. I have shown that the same lesion can be reproduced in dogs by the injection of bile into the duct, and have described a case in which a gall stone in the diverticulum of Vater had caused the penetration of bile into the pancreas, this producing the lesion. In the present case it is not possible to demonstrate such an irritant. Unfortunately it was not possible to perform a thorough autopsy. A point of some interest is the occurrence of hæmorrhagic vomiting after operation, suggesting the presence of some lesion of the stomach, possibly a perforating ulcer in contact with the pancreas, thus permitting contact of gastric juice with the parenchyma. The suggestion unsupported by other evidence has little value but emphasizes the importance of a more complete post-mortem examination in explaining the etiology of hæmorrhagic inflammation of the pancreas.

In connection with this case it is of interest to consider the frequency with which gall stones accompany acute hæmorrhagic pancreatitis. In two of the cases examined in the Pathological Laboratory, gall stones were present. It is difficult to obtain trustworthy figures from the literature, since in many cases the autopsy reports are so meagre that the presence or absence of cholelithasis cannot be determined. A considerable number of cases have been recently described by writers who have reported more than one case. Lund, of Boston, has reported five cases, three of which were examined after death, and in all of these gall stones were found. Bryant has reported two cases with gall stones. Hahn reports five cases, upon three of which an autopsy was performed; gall stones were found in one. Marx has described two cases with gall stones. Of these recently reported cases in which autopsies were obtained, in thirteen, including our three, gall stones were present in ten.

Since the diagnosis of acute lesions of the pancreas is difficult and at times impossible, the demonstration of a substance in the urine indicating such a condition would be important. Investigations of R. Langerhans and others have shown that disseminated fat necrosis accompanying

[1] Johns Hopkins Hospital Bulletin, 1901, Vol. XII, p. 184.

acute hæmorrhagic pancreatitis and other lesions of the pancreas is due to splitting of fat with the formation of fatty acid, and Flexner has demonstrated the presence of a fat splitting ferment in the areas of necrosis. It suggests itself that this ferment, which is free in the tissues may be excreted by the kidneys and for this reason I have made an examination of the urine in the present case. The results, though not conclusive, are worthy of attention, in order that similar examinations may be made in suitable cases. I used a method for the demonstration of fat splitting ferments, proposed by Prof. Castle and Mr. A. S. Loevenhart.[2] Ethyl butyrate, which is carefully purified, when acted upon by the ferment is decomposed with the formation of butyric acid whose presence is determined by litmus added to the solution.

Following the operation described the patient voided no urine and in the bladder at autopsy was found only a very small quantity. This was neutralized with potassium hydroxide and divided into two parts. To one part was added a few drops of ethyl butyrate together with a small quantity of litmus solution. The second part used as a control was boiled in order to destroy the ferment if present, and ethyl butyrate added. Both specimens were kept at 37° and at the end of twenty-four hours the unboiled specimen had acquired a well-marked acid reaction, while the control specimen was little if at all changed. Owing to the small quantity of urine obtained it was not possible to repeat the test which is described in order that its accuracy may be tested in other cases should the opportunity arise.

[2] American Chemical Journal, 1900, Vol. XXIV, No. 6.

Gonorrheal Peritonitis in a Child. Drs. Hunner and Harris.

(To appear later.)

NOTES ON NEW BOOKS.

Anatomy in its Relation to Art. By George McClellan, M. D., Professor of Anatomy at the Pennsylvania Academy of Fine Arts, etc., etc. (*Philadelphia: W. B. Saunders & Co., 1901.*)

This book of 142 pages with 125 plates which contain 338 illustrations, is intended to teach the artist the relation of the underlying structures to the surface of the human body, both in repose and in motion.

Anatomical knowledge is a very essential factor in the making of a true artist. In examining the annual output of the artistic profession of this country it becomes evident that the majority of painters and draughtsmen possess but vague ideas of the architecture of the human body.

The aid the camera affords has apparently been the factor causing the artist to partly abandon the excellent practice of the old masters, who studied by drawing free-hand from the model and the cadaver, thus determining in every portion of the body the causes producing the manifold characteristic forms. The camera undoubtedly has brought about a greater accuracy as to the contour and done away with many mannerisms of the old school, but it has also introduced a general indifference among artists when dealing with anatomical facts.

It is universally considered, that the most objective interpretation of nature is the best, because the artist's individuality is then least perceptible. This excuse is offered by those who wish to evade the trouble of studying anatomy; they say: "if one knows the causes for all the peculiar forms characteristic of the human body, one is very apt to overdo the detail work and represent nature not as she appears to our eye, but as our trained mind knows her to be." Then they quote great masters and great teachers and advise us to simply close one eye so as to do away with the confusing stereoscopic picture seen with two sound eyes and then proceed to copy nature "naïvely" and faithfully.

It is true that noble simplicity of form and outline has often been sacrificed for an exaggerated truthful anatomical representation of the human body; in other words, Beauty and Poetry are absent where the mathematical mind rules.

But this is not necessarily the case. The knowledge of anatomy in art is as essential for the proper representation of beauty in thought and conception as thorough bass is in music and grammar in literature. To be sure, the anatomical knowledge of the artist should not be so much in evidence as to force itself upon the observer. All the landmarks of the bony framework of a figure should be in their proper places, though not all visible, and the muscles and tendons correctly attached, but there should be no exaggeration in detail, no muscle or fascia should be indicated unless it leaves an unmistakable protuberance or depression on the skin surface.

When studying from life, the artist is impressed with the great number of folds and depressions on the skin surface, some of which he presumes are constant, while others just happen to be there in his particular model. Clear anatomical knowledge will in such cases enable him to decide what is correct and typical, and therefore beautiful, and what should be regarded as an imperfection in the model. He knows at once how to discriminate between what ought to be imitated and studied and what discarded. Many of the best known artists of the past and of the present day must have had a vague conception of the healthy and normal human body, for they have often pictured figures as types of beauty or as their own ideals which showed slight, though unmistakable signs of disease, abnormal development or acquired deformities.

Every effort to teach anatomy to the artist is therefore a very commendable undertaking. There are many books on the subject, none of which are perfect. Those published by artists are generally worthless from an anatomical standpoint, while those written and illustrated by medical men give as a rule wretched pictures. It is true, that the artist can study anatomy better from the latter than from the former, but the teaching is by no means perfect. The bones, fascia and muscles composing a body, are not devoid of beauty in an artistic sense, and to portray them correctly a high degree of art is necessary, certainly more than one can expect of a pure scientist. The outlines of the individual bones for instance possess a characteristic grace, which the artist should be taught to see and appreciate, but if a book on anatomy pictures bones like pieces of rubber tubing joined together, the student must necessarily experience considerable difficulty in arriving at an understanding of the beauty of the human body.

The present contribution to our literature on Anatomy in

Art is a meritorious presentation of the subject; though it has the above-mentioned shortcomings. The author says in his preface that he "makes no pretension to enter the field of an artist, as it is only claimed that the drawings are diagrammatic expositions of the anatomy of the human body, where photographs would not serve the purpose," etc. These drawings are indeed very crude, and while they teach anatomy no better than it can be found in any of the standard text-books, their shapeless forms must have an undesirable effect upon the beginning art student. A book that is to analyze forms should at least picture them faithfully and with a suggestion of the three dimensions, even though only outlines be used, for a well-drawn contour is capable of indicating a form almost as correctly as an elaborately shaded picture.

The book contains not only drawings but also reproductions of photographs of two models, a male and a female, in different poses, which are quite instructive. They would be more so if the detail showed more plainly, a deficiency due probably to imperfect illumination of the model. As it is, the majority of these photographs appear more as grey silhouettes than plastic bodies. Moreover, the models employed show a number of deficiencies of form, which should have been especially mentioned in the legends, so as to avoid the risk of impressing imperfect forms upon the memory of the student.

The best pictures and the most instructive are three views of Houdon's famous statue "L'écorché"; the accompanying three poses of the male model used in the book, which are in the same position as the statue, seem superfluous. The same can be said concerning other similar poses, although as said above, a number are quite useful and a full-fledged artist, having the gift of discrimination will undoubtedly enjoy studying them.

The text is clear and concise and it gives much that is interesting to both artist and anatomist. The different chapters are short. After an introductory note we find descriptions of: the skeleton—articulations—bones of head and face, muscles of head and face—the neck—the trunk, thorax, abdomen, back—the upper extremity—the pelvis—the lower extremity—general considerations of the entire skeleton—influence of the skeleton on the surface form—influence of the muscles on the surface form—attitudes and postures—movements of progression—proportions of the human figure.

The book is rendered voluminous by the great number of plates, while the text is so concise, that it may be read in one sitting. The book is well made and aside from the fact that the text is printed in gray, the pages look well.

Gynæcological Pathology. A Manual of Microscopical Technique and Diagnosis in Gynæcological Practice for Students and Physicians. By DR. CARL ABEL, Privat-Docent in Berlin. Translated and edited by Samuel Wyllis Bandler, M. D., Adjunct Gynæcologist of the Beth Israel Hospital, New York, with a chapter on the Embryology of the Female Genitalia and the Pathological Growths developing from Embryonal Structures. Illustrated by 100 engravings. (*New York: William Wood & Co., 1901.*)

The first chapter is devoted to the method of obtaining uterine tissue for diagnosis. In the first place we would mention that if the speculum advocated be used perfect asepsis is almost out of the question, as we are advised to fasten the speculum to the table. Then again when pieces of cervix are examined the bleeding is usually checked by packing with cotton; where any appreciable amount of tissue is removed it is always better to approximate accurately with sutures instead of leaving the tissue to granulate. We are pleased to note the careful macroscopical examination of scrapings and we are advised to make frequent use of the magnifying glass. Many cases of malignant growth may undoubtedly be recognized in this manner. The various methods of cutting and staining are briefly and yet clearly given. Abel finds it necessary to leave a uterus in Müller's fluid from six to eight weeks before sufficient hardening has taken place. If he placed the specimen in the thermostat and changed the fluid whenever it became cloudy less than two weeks would be the requisite period. His remarks on so-called erosions of the cervix and also on the eversions of the cervical mucosa are very clear and to the point. The confusing and suspicious pictures occasioned by the irregular and yet benign dippings down of the normal cervical epithelium are carefully dealt with. Squamous-cell carcinoma and adeno-carcinoma of the cervix are, however, not very clearly depicted and from them the student will gain but a hazy idea of the various stages of the disease. Hyperplasia or as we are accustomed to term it hypertrophy of the mucosa is well described and suitably illustrated. But cancer of the body is allowed but little space, although it is exceedingly important. In summing up the description of carcinoma of the body of the uterus Abel says "the deep extension of the glandular neoplasm into the muscle is then the only criterion of the malignancy of the neoplasm." Were the author right in his assumption we would be in a sorry plight. In the early stages of the disease the curettings rarely yield muscle and it is only in the fairly advanced and late stages that muscle is found in the tissue removed by the curette. Abel directs little or no attention to the atypical gland grouping and to the pathological changes in the individual cells. These are the true factors in the diagnosis and from them alone the diagnosis can be made in nearly every case. Of course if muscle be present we have additional confirmatory evidence but this is not at all necessary. Uterine myomata and also dermoid cysts are treated in a very cursory manner, the former not being accorded even one page. Inflammations of the tube are fully and very satisfactorily described and the description of tubal pregnancy is undoubtedly the best in the book. The anatomy of the ovary is very clear but the descriptions of pathological conditions meagre. Abel mentions having found small cyst-like spaces lined by giant cells in a dermoid cyst of the ovary. Little attention has been paid to this in the past. We have found them repeatedly in dermoid cysts. In fact, they are undoubtedly pathognomonic of dermoids and whenever we have found them in the ovary further sections revealed the presence of a dermoid no matter how small the cyst was.

Undoubtedly the most interesting portion of the book is Part III, which has been added by the translator. Bandler has given us a very clear idea of the embryology of the female genitalia and has been most judicious in his selection of illustrations from the various monographs on the subject. He has also given us a full survey of the pathological growths developing from the embryological structures, paying especial attention to adeno-myomata.

The text on the whole is clear, but in several places some obscurity exists, for example, on page 67: "At times there develops in a carcinoma an adenoma above the remaining epithelium of the cervical mucous membrane, i. e., the glands so that a mixed tumor results (adeno-sarcoma)." We presume that the word carcinoma instead of sarcoma has been accidentally inserted, but even then it will be rather difficult for a skilled pathologist to get his bearings.

The illustrations in part are very good, but many, while presenting a general idea of the histological picture, on careful scrutiny fail to give the finer details so necessary for the student.

The Four Epochs of Woman's Life. A study in Hygiene. By ANNA M. GALBRAITH, M. D., Fellow of the New York Academy of Medicine, etc. With an Introductory Note by JOHN H. MUSSER, M. D., University of Pennsylvania. 12mo. volume of 200 pages. (*Philadelphia and London: W. B. Saunders & Co., 1901.* Cloth, $1.25, net.)

This book is written by a woman physician for women who are supposed to be unacquainted with the essential facts of their sexual life. It seems upon the whole judiciously written, although there is at times too much technical knowledge for the unscientific reader and too little for the thorough student. A tendency is noticeable which is not altogether absent from similar semi-scientific and semi-popular works written by women to enter upon discussions which have but a remote, if any, connection with the subject-matter of the book. Thus the present book commences with a brief essay upon education and the correlation of mind and body. The views presented are unimpeachable and in fact are derived from standard authors, but they seem out of place in a treatise whose avowed object it to teach woman about her sexual system. The book otherwise is clearly and properly written and seems well calculated to instruct young women, especially young wives and mothers in matters which they ought to know.

First Aid to the Injured and Sick. By F. J. WARWICK, B. A., M. B. Cantab., Associate of King's College, London; Surgeon-Captain, etc.; and A. C. Tunstall, M. D., F. R. C. S. Ed., Surgeon-Captain, etc. 16mo volume of 232 pages and nearly 200 illustrations. (*Philadelphia and London: W. B. Saunders & Co., 1901.* Cloth, $1.00 net.)

This compact, handy and useful manual can be recommended to all who are called upon to meet sudden accidents or emergencies. The chapters on bandaging, the immediate treatment of hemorrhage, artificial respiration and the transportation of the wounded are especially valuable. The directions given are clear and easily understood.

Outlines of Anatomy. A guide to the methodical study of the human body in the dissecting room. By EDMUND W. HOLMES, A. B., M. D., Demonstrator of Anatomy, University of Pennsylvania, etc. Second edition. (*1902, Press of the New Era Printing Co., Lancaster, Pa.*)

The intention of this manual of less than two hundred pages is to introduce method and discipline into the dissecting room and to awaken an interest in practical anatomy. The course of dissection laid down is designed to consume twenty-eight days, which would seem to be a minimum requirement for practical anatomy. The book is good but elementary and the impression which it leaves upon the reader is that the author in his teaching sought to teach anatomy rather than to make anatomists. The purpose of the book is excellent even if the standard is not high.

Essentials of Physiology. Prepared especially for Students of Medicine; and arranged with questions following each chapter. By SIDNEY P. BUDGETT, M. D., Professor of Physiology, Washington University, St. Louis. 16mo volume of 233 pages, illustrated. (*Philadelphia and London: W. B. Saunders & Co., 1901.* Cloth, $1.00 net.)

An excellent and probably the distinguishing feature of this compend is the chapter on the nervous system which is fully illustrated by half-tone reproductions of schematic representations of various parts of the brain and spinal cord. The book as a whole is marked by clearness and conciseness of statement and if one needs a question compend this may be recommended as a reliable and modern guide.

BOOKS RECEIVED.

A Text-Book of Surgery. By Dr. Hermann Tillmanns. Translated from the seventh German edition by Benjamin T. Tilton, M. D., and John Rogers, M. D. Edited by Lewis A. Stimson, M. D. Volume I. The Principles of Surgery and Surgical Pathology. With five hundred and sixteen illustrations. 1901. 8vo. 841 pages. D. Appleton and Company, New York.

A Treatise on the Acute, Infectious Exanthemata. Including Variola, Rubeola, Scarlatina, Rubella, Varicella, and Vaccinia, with especial reference to Diagnosis and Treatment. By William Thomas Corlett, M. D., L. R. C. P. Lond. Illustrated by 12 colored plates. 28 half-tone plates from life and 2 engravings. 1901. 8vo. 392 pages. F. A. Davis Company, Philadelphia.

A Text-Book of Physiological Chemistry. For Students of Medicine and Physicians. By Charles E. Simon, M. D., 1901. 8vo. 453 pages. Lea Brothers and Company, Philadelphia and New York.

Twenty-eighth Annual Report of the Secretary of the State Board of Health of the State of Michigan. For the fiscal year ending June 30, 1900. 8vo. XXVII + 272 pages. 1901. Lansing, Michigan.

Transactions of the State Medical Association of Texas. Thirty-third annual session held at Galveston, Texas, April 23rd to 26th, 1901. 8vo. 463 pages. Austin, Texas.

Transactions of the Clinical Society of London. Volume the thiry-fourth. 1901. 8vo. lx+270 pages. Longmans, Green, and Company, London.

Transactions of the American Surgical Association. Volume the nineteenth. Edited by Richard H. Harte, M. D. 1901. 8vo. xxviii+514 pages. Printed for the Association. Philadelphia.

Water and Water Supplies. By John C. Thresh, D. S. C. (London); M. D. (Victoria); D. P. H. (Cambridge). Third edition, revised and enlarged. 1901. 12mo. 527 pages. P. Blakiston's Son and Company, Philadelphia.

Laboratory Work in Histology. By G. Carl Huber, M. D. Third edition, revised and enlarged. [1901.] 8vo. 204 pages. George Wahr, Ann Arbor, Michigan.

Epilepsy and Other Chronic Convulsive Diseases. Their Causes, Symptoms and Treatment. By Sir William R. Gowers, M. D., F. R. C. P., F. R. S. Second edition. 1901. 8vo. xxiv + 320 pages. P. Blakiston's Son and Company, Philadelphia.

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